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Nineteen Thirty-Five

IN extending the conventional greeting of the season to the veterinary profession we find reason for congratulations, not only for the clinical branch which has passed a trying period without loss of spirit or prestige, but also to the other branches into which the application of veterinary medicine is divided;

to the veterinary profession as a whole for the brilliant page written into the history of American veterinary medicine in having conducted the Twelfth International Veterinary Congress with indelible credit to the veterinarians of the United States;

to the field of research and investigation for the progress it has made in the diagnosis and control of Bang's disease without which the program now in the course of execution would have been impossible;

to the regulatory veterinary service of the Federal Government and of the States for bringing the colossal undertaking of eradicating bovine tuberculosis a long step nearer to its ultimate objective;

to the clinical branch for its qualification to render the service required to carry out these projects, and the spirit of cooperation displayed;

to the literary branch for the excellent contributions to the literature published, and for the constructive policies advocated in behalf of the veterinary service and live stock industry.

The aim for nineteen thirty-five continues the policy of "selling satisfaction" to graduates of recognized veterinary colleges only, in the form of pace-making products in the biological, pharmaceutical, and surgical fields, through our eighteen branches located conveniently throughout the territory of the Middlewest.

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H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

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AN ENCOURAGING OUTLOOK

With so many things out of joint nowadays, it was perfectly natural for someone to ask the question, "Why should the number of veterinary students be increasing when the number of animals in the country is decreasing?" A fair question and one that involves many considerations, as the implication is that there should be a direct ratio between the animal population of the country and the number of veterinarians.

In the first place, the activities of veterinarians have become so diversified that there is no longer any direct ratio between the number of animals and the number of veterinarians. One factor alone, the automobile, has put an entirely different aspect on this particular question. Today a veterinarian can cover several times the amount of territory that could be covered in the day of the horse and buggy. This argues for fewer veterinarians. However, in many localities there are more animals and more problems for the veterinarian today than there were a generation ago. Whereas, thirty years ago the practitioner occupied himself almost exclusively with curing the sick and repairing the lame and the injured, today he interests himself in applying large-scale methods of prophylaxis and systems of eradication, as far as the serious infectious diseases of animals are concerned. The picture of general practice has almost completely changed.

But, to return to the question. We have very distinct recollections of the recent pig-killing campaign. Without discussing the merits or the demerits of the plan, it did seem rather repulsive, rather disconcerting, to kill deliberately five or six million pigs, after years of study, research and effort by veterinarians and animal husbandmen designed to enable farmers and breeders to produce and save more pigs. The most recent estimates indicate a reduction of about one-third in the combined spring and fall pig crop for 1934 compared with 1933, and the prospect of 17 per cent fewer sows due to farrow this spring, compared with the spring of 1934, which was considerably below average.

Close on the heels of the pig slaughter of late 1933 came the announcement of the plan to combat Bang's disease in a big way. Theoretically, if all cattle in the United States are tested and the reactors slaughtered, this will mean a reduction of 15 per cent in the number of cattle in the country. Actually, this will not be the case, as all cattle will not be tested. However, there are very good prospects for a marked reduction in the cattle population, particularly in view of increased activities in the tuberculosis-eradication campaign, and the inclusion of mastitis in the AAA program.

Reports from western points indicate that the available supply of sheep is 25 per cent below a year ago, with the lamb crop running 40 to 50 per cent less than one year ago. And then, with almost a suggestion of mockery, because there had not been any concerted effort to reduce the number of horses, along comes that old nightmare of veterinarians, so called "corn-stalk disease," to take off tens of thousands of horses in a couple of months in the Central West, just at a time when the horse was beginning to stage a real come-back. Add to this the fact that farmers have been disposing of their poultry at an alarming rate because of the high costs of feed and the relatively low prices for eggs. On the whole, apparently not a very pleasant prospect for veterinarians.

But let us look beneath the surface. There are certain inexorable laws still operating. Several of these are intimately concerned with two factors, supply and demand. It is reasonably safe to figure that the reduction in the number of animals will result in increasing the value of those that are left. Of course, this has been one of the principal objects of the purely artificial measures put forth to reduce the number of swine and cattle. But let us look even a bit further. The cattle that have been removed have been, not only theoretically but actually, the least

valuable. Many have been inferior specimens by reason of the fact that they were suffering from either tuberculosis, Bang's disease or mastitis. By removing a large number of cattle from the lower price brackets, the average of those remaining has been given a considerable boost. Concurrently two factors thus have been operating to move prices in the same direction—upwards.

The net result of all this unusual disturbance in animal economics can hardly be anything else than an appreciable increase in the value of farm animals for the next several years. With each animal worth more money, owners naturally will take better care of them, and here is exactly where the veterinarian steps prominently into the picture. There should be a very noticeable increase in the demand for veterinary services and, furthermore, there should be a better ability upon the part of farmers and breeders to pay for these services.

With the elimination of so many of the unfit, it is to be assumed that those animals remaining will enjoy a better state of health, greater freedom from disease, higher productivity, increased capacity for reproduction, in fact, all of those qualities which combine to produce a better state of well-being among our herds and flocks. After all, is this not the real goal of veterinary science? Is not the true function of the modern veterinarian that of the animal engineer? Is it not more pleasant to work among healthy animals than among those that are diseased? What more discouraging task than to be called upon to treat a herd of hogs in which cholera has made its appearance. And how unnecessary!

There is just one fly in the ointment. Owners of animals will continue to act like human beings. Freedom from animal diseases may propagate an inherent tendency to take chances. Owners of animals may not feel the urge to employ veterinarians to hold disease down to a low point, once it is down. A false sense of well-being may breed carelessness. It often does, not only with disease, but with other hazards. Human frailty, after all.

On the other hand, cattle, hogs, horses, sheep and poultry are tangible property. In the main, they are cash crops. They are not sold on time, like many products of the factory, such as automobiles, radios, jewelry and clothing. Even though the farmer has had the reputation of being a poor business man, and has been slow to change his ways, he has had numerous opportunities lately to know more about veterinarians and their work. It should not be difficult for him to figure out a direct connection between preventive veterinary medicine and his own success with his breeding and feeding operations. Let us hope that he will

figure this out for himself before somebody does it for him in a way that he may not like. If he does, there will be plenty of work for veterinarians, and work that will be pleasant and very much worth while.

EXTENDED ITINERARY FOR PRESIDENT MACKELLAR

Another president of the American Veterinary Medical Association is finding out what really goes with this office. A few years ago, within three months of the time he was elected to the office, an A. V. M. A. president remarked that he had just about decided that he had become a traveling man, for a year at least. It is a very encouraging sign when so many veterinary associations in all parts of the country indicate a desire to have a visit from the president of the A. V. M. A., and it is very much to be regretted that it is never possible for an A. V. M. A. president to accept all of the invitations which he does receive. There are two limiting factors. The first of these is the inescapable conflict of dates, particularly during the winter months when many of the state associations hold their annual meetings. It is just a physical impossibilty for one person to be in two or more places at the same time. The second factor is the necessary limitation of the funds available for the traveling expenses of the president.

Dr. MacKellar got off to an early start and attended three large meetings and several smaller ones before the end of October rolled around. The larger meetings including the fifth annual meeting of the New England Veterinary Medical Association, in Boston, Mass., October 22-23; later in the same week, he attended the 52nd annual meeting of the Pennsylvania State Veterinary Medical Association, in Scranton; the following week saw him in Jacksonville, Fla., to attend the 19th annual meeting of the Southern States Veterinary Medical Association, held in conjunction with the annual meeting of the Florida State Veterinary Medical Association. In attending these three meetings, President MacKellar was able to pay his respects to the six New England states, Pennsylvania and ten southern states.

The first week in December, President MacKellar was in Chicago attending the semi-annual meeting of the Executive Board of the A. V. M. A., the 38th annual meeting of the United States Live Stock Sanitary Association and the annual conclave of the National Association of B. A. I. Veterinarians. At the Executive Board meeting, an itinerary for the balance of the year

was approved, and this will make it possible for President MacKellar to attend at least eight more meetings. During the first week of the present month, he will attend the annual meeting of the Veterinary Medical Association of New Jersey, in Trenton; the following week, he will proceed to Philadelphia to attend the 35th annual Conference of Veterinarians, at the University of Pennsylvania; he will then proceed to Ithaca, N. Y., to participate in the 27th annual Conference for Veterinarians at Cornell University. The third week in January will see President MacKellar in Columbus to attend the 52nd annual meeting of the Ohio State Veterinary Medical Association. The following week, he will go to Des Moines for the first day of the annual meeting of the Iowa Veterinary Medical Association. He will have to cut short his stay in Iowa in order to get back to East Lansing, Mich., for the latter part of the program of the Michigan State College Short Course for Veterinarians.

One meeting is scheduled for February, that of the Maryland State Veterinary Medical Association, in Baltimore, on the 7th. Then, there is quite a jump to July 10, 11 and 12, the dates scheduled for a joint meeting of the veterinary medical associations of Virginia, North Carolina, Maryland and the District of Columbia, in Richmond, Va. In all probability, Dr. MacKellar will find it possible to attend meetings of other associations close to New York, of which there are quite a number. When it is considered that Dr. MacKellar is engaged in general practice, it will be appreciated how much of a sacrifice he is making this year in attending to his official duties as president of the A. V. M. A., involving, as this does, many days of absence from his practice.

MICHIGAN AND WASHINGTON IN LINE

Shortly after the announcement was made that the University of Pennsylvania had decided to adopt the five-year course in veterinary medicine (see December JOURNAL), a letter was received from Dean Giltner, of Michigan State College, stating that that institution would require a year of college work for entrance to the veterinary course in the fall of 1935. While we were considering the probable effects of this step and wondering which of the remaining four-year colleges would be the next to make this move, word reached us from Dean Wegner to the effect that the State College of Washington had joined the procession, making eight in line. Which institution will be next? Come on, Alabama, Ontario and Texas, let's make it unanimous.

CONVENTION DATES FIXED

At the meeting of the Executive Board held in Chicago on December 5, 1934, the dates for the Oklahoma City convention were fixed for August 27, 28, 29 and 30, 1935, these dates having been recommended by the Oklahoma veterinarians, through their spokesman, Dr. C. C. Hisel, of Oklahoma City. Watch the JOURNAL for further announcements concerning the selection of official headquarters, organization of the Committee on Local Arrangements and other matters pertaining to the convention.

VISITORS AT THE JOURNAL OFFICE

The past month saw quite a number of prominent members of the A. V. M. A. at the association headquarters in Chicago on various missions. The list included: Dr. Cassius Way, of New York, chairman of the Executive Board; Dr. R. S. MacKellar, of New York, president of the A. V. M. A.; Dr. M. Jacob, of Knoxville, Tenn., treasurer of the A. V. M. A.; Dr. F. F. Parker, of Des Moines, Iowa, and Dr. L. A. Merillat, of Chicago, members of the Executive Board; Dr. C. H. Stange, of Ames, Iowa, and Dr. N. S. Mayo, of Highland Park, Ill., members of the A. V. M. A. Committee on Education; Dr. Ward Giltner, of East Lansing, Mich., A. V. M. A. Representative to the American Association for the Advancement of Science; Dr. John R. Mohler, of Washington, D. C., and Dr. A. Eichhorn, of Pearl River, N. Y., on International Veterinary Congress business; Dr. M. W. Emmel, of Gainesville, Fla.; Dr. I. D. Wilson, of Blacksburg, Va.; Dr. Edward Records, of Reno, Nev., newly elected president of the U. S. Live Stock Sanitary Association; Dr. R. L. Conklin, of MacDonald College, Quebec; Dr. E. C. W. Schubel, of Blissfield, Mich., secretary of the Michigan-Ohio Veterinary Medical Association; Dr. C. M. Carpenter, of Los Angeles, Calif., secretary of the California State Veterinary Medical Association, accompanied by Mrs. Carpenter, who has very kindly offered to paint the portraits of the presidents of the A. V. M. A.

APPLICATIONS FOR MEMBERSHIP

This month we are listing eleven applications for membership, the largest number to be given first listing in January since 1931. The applications listed for the first time in January are those

received during the month of December, usually an off month. During the twelve months of 1934, there were 112 applications listed, this total exceeding the number of applications received for each of the two years preceding. These gains, although not very large, are encouraging. Quite a few inquiries have been received recently from former members, dropped for the non-payment of dues, asking how they could secure reinstatement and, in this connection, it might be well to point out here that there are two ways by which former members of the A. V. M. A. may be placed back on the roll: (1) by paying all arrearages or (2) filing a new application for membership.

Owing to the fact that the By-laws provide for carrying a member for two full years before dropping him for the non-payment of dues, such a member owes at least $10 for dues when he is dropped. As a matter of fact, the actual removal of his name from the roll usually takes place during the third year of his delinquency and, by that time, the member owes dues for three years, or $15. These facts are given in explaining one of the ways by which reinstatement may be secured, namely, the payment of arrearages. The other way is to file an application and join all over again, just the same as would be the case with a veterinarian who had never been a member of the A. V. M. A. It will be appreciated that the latter method is invariably less expensive and is the way usually recommended. It is the slower of the two ways, as an application cannot be completed in less than 60 days. On the other hand, reinstatement can be secured immediately by the payment of back dues and without the formality of filing an application. Members who have resigned in good standing may be reinstated only by making reapplication for membership.

As is customary, at intervals of six months we publish that section of the By-laws describing the manner of filing an application for membership. Note particularly the provisions for posting the names of applicants for a period of 60 days (two consecutive issues of the JOURNAL). Members should scan the list of applicants each month and see just who is applying for admission to the A. V. M. A. Just a short time ago, a member expressed considerable surprise upon finding out that another veterinarian was a member of the A. V. M. A. There had been a little trouble of some kind involving the other veterinarian and the inquiring member was advised that the application of his colleague had been filed and listed in the regular manner prescribed by the By-laws and, in the absence of any objections, the applicant had been admitted to membership.

Section 1 of Article 2 of the By-laws reads as follows:

Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BARBER, FRED A. Box 207, The Dalles, Ore.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: L. C. Henderson and Robert Jay.

BINNIG, JUDSON H. Millersburg, Ohio
D. V. M., Ohio State University, 1934
Vouchers: Oscar V. Brumley and James D. Grossman

COCKING, ROGER M. Iowa State College, Ames, Iowa
D. V. M., Iowa State College, 1934
Vouchers: H. L. Foust and T. S. Leith.

FREDERICK, LAURENCE D. Swift & Company, Chicago, Ill.
D. V. M., Cincinnati Veterinary College, 1918
Vouchers: L. A. Merillat and H. Preston Hoskins.

MANGONON, PATRICIO S. Nanning, Kwangsi, China
D. V. M., University of the Philippines, 1932
Vouchers: E. A. Rodier and Greg. San Agustin.

MARKHAM, FRED D. Lincoln St., Port Leyden, N. Y.
M. D. C., Chicago Veterinary College, 1895
V. S., Ontario Veterinary College, 1905
Vouchers: W. J. Sellman and Don A. Boardman.

ROBNETT, ERNI V. 811 South Walker, Oklahoma City, Okla.
D. V. S., Kansas City Veterinary College, 1902
Vouchers: L. J. Allen and C. H. Fauks.

SEWELL, KENNETH D. 1541 Kingsway, Vancouver, B. C., Can.
B. V. Sc., Ontario Veterinary College, 1914
Vouchers: R. G. Cuthbert and J. G. Jervis.

THOMPSON, EDWARD E. Woodstock, Va.
D. V. M., Iowa State College, 1934
Vouchers: I. D. Wilson, E. P. Johnson and R. A. Runnells.

THORPE, MARVIN S. 6055 Gwynn Oak, Baltimore, Md.
B. S., D. V. M., State College of Washington, 1933
Vouchers: J. S. Koen and E. P. Yager.

WALKER, EARL M. Box 327, Redfield, S. Dak.
B. S., D. V. M., Iowa State College, 1934
Vouchers: G. E. Melody and E. E. Flory.

Applications Pending

SECOND LISTING

(See December, 1934, JOURNAL)

Bigelow, Balfour, 531 E. Olive St., Turlock, Calif.
Dennis, William A., 9 Elmwood Ave., Jamestown, N. Y.
Elwood, Guilford S., 1904 W. North Ave., Chicago, Ill.
Floyd, James H., Pepper Ridge Rd., Stamford, Conn.
Neal, Chas. W., 1619 S. Laredo St., San Antonio, Texas.

The amount which should accompany an application filed this month is $10.00, which covers membership fee and dues to January 1, 1936, including subscription to the JOURNAL.

Dogs Demonstrate Devotion

History is full of examples of the blind, unswerving devotion that dogs have given their masters and mistresses through the ages. Nor is this devotion reserved for any one breed of dogs, according to *Animaldom*, which cites two classic examples.

On the morning of the execution of Mary, Queen of Scots, her Skye terrier was found to be missing. When the blood-stained body of the unfortunate queen was taken up, the dog was found crouching beneath her robes. He would not depart. A lady took charge of him, but he refused all further connection with human beings. Then, there is the Pekingese which stood over the bodies of the late Tsar of Russia and his family and challenged the assassins until a bullet settled his brave defiance forever.

STATE BOARD EXAMINATIONS

Kansas State Board of Veterinary Examiners. State House, Topeka, Kan. January 8, 1935. Dr. Thos. P. Crispell, Secretary, Parsons, Kan.

Oklahoma State Board of Veterinary Medical Examiners. Capitol Building, Oklahoma City, Okla. Regular examination, January 16, 1935. Any veterinarian wishing to qualify should report at the State Veterinarian's office, at 8:30 a. m., on above date. Information and application blanks may be obtained from Dr. Walter H. Martin, Secretary, 101 S. Evans St., El Reno, Okla.

COMING VETERINARY MEETINGS

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. January 2, 1935. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. January 2, 1935. Dr. Harley B. Wood, Secretary, 2754 Meramec St., Saint Louis, Mo.

Minnesota State Veterinary Medical Society. Hotel Lowry, Saint Paul, Minn. January 3-4, 1935. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

New Jersey, Veterinary Medical Association of. Hotel Hildebrecht, Trenton, N. J. January 3-4, 1935. Dr. John G. Hardenbergh, Secretary, care Walker-Gordon Lab. Co., Plainsboro, N. J.

Delaware Veterinary Medical Association. University of Delaware, Newark, Del. January 4, 1935. Dr. J. R. West, Secretary, 219 S. Walnut, Milford, Del.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 4-5, 1935. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 7-10, 1935. Dr. Cliff D. Carpenter, Secretary, 337 Central Ave., Los Angeles, Calif.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. January 8, 1935. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Northwestern Missouri Veterinary Medical Association. Maryville, Mo. January 8, 1935. Dr. E. S. Harrison, Secretary, Burlington Junction, Mo.

Rhode Island Veterinary Medical Association. State House, Providence, R. I. January 8, 1935. Dr. J. S. Barber, Secretary, 14 Washington St., Central Falls, R. I.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 8-9, 1935. Dr. C. J. Marshall, Chairman, 39th St. and Woodland Ave., Philadelphia, Pa.

Maine Veterinary Medical Association. Augusta, Maine. January 9, 1935. Dr. R. E. Libby, Secretary, Richmond, Me.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. January 9, 1935. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Western Michigan Veterinary Medical Association. Grand Rapids, Mich. January 10, 1935. Dr. C. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 10-11, 1935. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 14-15, 1935. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

Interstate Veterinary Medical Association. Elks Building, Omaha, Nebr. January 14, 1935. Dr. G. L. Taylor, Secretary, Plattsmouth, Nebr.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. January 15, 1935. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Kansas Veterinary Medical Association. Topeka, Kan. January 16-17, 1935. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Ohio State Veterinary Medical Association. Columbus, Ohio. January 16-17, 1935. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Colorado Veterinary Medical Association. Albany Hotel, Denver, Colo. January 17, 1935. Dr. J. C. Flint, Secretary, Colorado Agricultural College, Fort Collins, Colo.

Tennessee Veterinary Medical Association. Nashville, Tenn. January 21-22, 1935. Dr. A. C. Topmiller, Secretary, Department of Agriculture, Nashville, Tenn.

Wisconsin Veterinary Medical Association. Madison, Wis. January 21-23, 1935. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 21-25, 1935. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 22-24, 1935. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Iowa Veterinary Medical Association. Fort Des Moines Hotel, Des Moines, Iowa. January 22-24, 1935. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Keystone Veterinary Medical Association. Philadelphia, Pa. January 23, 1935. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 29-31, 1935. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

Nevada State Veterinary Association. Reno, Nev. January 30, 1935. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 4-9, 1935. Dr. C. A. Cary, Dean, Alabama Polytechnic Institute, Auburn, Ala.

Connecticut Veterinary Medical Association. Hotel Bond, Hartford, Conn. February 6, 1935. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

Maryland State Veterinary Medical Association. Medical Hall, Baltimore, Md. February 7, 1935. Dr. Mark Welsh, Secretary, College Park, Md.

Hudson Valley Veterinary Medical Society. Albany, N. Y. February 13, 1935. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Illinois State Veterinary Medical Association. Leland Hotel, Springfield, Ill. February 19-20, 1935. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

Northwestern Ohio Veterinary Medical Association. Hotel Waldorf, Toledo, Ohio. February 20, 1935. Dr. W. P. S. Hall, Secretary, Division of Health, 9 Ontario St., Toledo, Ohio.

Louisiana Veterinary Medical Association and Louisiana State University Veterinary Short Course. Dalrymple Memorial Building, Louisiana State University, Baton Rouge, La. March 6-7, 1935. Dr. H. A. Burton, Secretary, 1506 Park Ave., Alexandria, La.

We are more sensible of what is done against custom than against Nature.—PLUTARCH.

THE ACID-BASE BALANCE IN COWS AND EWES DURING AND AFTER PREGNANCY, WITH SPECIAL REFERENCE TO MILK FEVER AND ACETONEMIA*

By JESSE SAMPSON *and* C. E. HAYDEN

Department of Veterinary Physiology
New York State Veterinary College, Cornell University
Ithaca, N. Y.

This report is based on studies of the acid-base balance in normal and abnormal cows and ewes, with special reference to pregnancy and to pre- and post-parturient disorders. Quantitative determinations have been made of the CO_2-combining power of the blood plasma (alkali reserve), serum calcium, total blood ketones, and, to a limited extent, of the total ketones and ammonia nitrogen of the urine.

The data that have been accumulated are not so comprehensive as had been hoped for; hence, further studies of the acid-base balance in cattle and sheep are desirable. Nevertheless, the results reported in this paper are suggestive of a number of fairly well defined conclusions.

The case reports of the various diseases studied have been omitted purposely; the diagnoses, however, were made by skilled clinicians.

A number of investigators—among them, Allen,[1] Allen and Wishart,[2] in a series of experiments with dogs; Levine and Smith,[3] using rats; Carpenter,[4] Blatherwick,[5] Hart, Steenbock and Humphrey,[6] Robinson and Huffman,[7] and Perkins, Hayden and Monroe,[8] studying cattle; and Forbes, Halverson and Schulz,[9] working with swine—have published some interesting results of studies that have either a direct or indirect bearing on the general subject of acidosis in mammals. There is evidence, in several of the reports based on these studies, that the alkali reserve of the blood, in some species, can be significantly increased or decreased by feeding rations that are either potentially acid or basic in character. A number of these studies have demonstrated, also, that ketosis and acidosis are not readily produced in domestic animals by certain experimental methods, *e. g.*, fasting or by the feeding of unbalanced rations.

*Abstract of a thesis by Jesse Sampson submitted to the faculty of Cornell University in partial fulfillment of the requirements for the degree of Doctor of Philosophy, September, 1933.

I. Results of the Studies with Cows

During and After Pregnancy

In the studies with cows, an attempt first was made to determine whether there were any characteristic changes in the blood and urine associated with pregnancy and during the first three days after parturition. (Milk fever in cows is said rarely to occur beyond the first three days after calving.[10]) In a number of instances, additional samples of blood and urine were taken at short intervals up to three to six weeks after the termination of pregnancy.

Blood and urine analyses: CO_2 capacity of plasma—70 determinations on 13 pregnant cows, range 56.7 to 79.6 vol. per cent, av. 62.34; 42 determinations on 12 cows after parturition, range 53.8 to 70.0 vol. per cent, av. 64.32. Serum calcium—60 determinations on 13 pregnant cows, range 10.23 to 12.50 mg per 100 cc, av. 11.18; 46 determinations on 12 cows after parturition, range 9.26 to 11.08 mg per 100 cc, av. 10.01. Total blood ketones*—13 determinations on 9 pregnant cows in late pregnancy, range 2.09 to 4.72 mg per 100 cc, av. 3.30; 44 determinations on 13 cows after parturition, normal range 1.87 to 5.51 mg per 100 cc, av. 2.93. Total urine ketones—10 determinations on 7 cows after parturition, normal range 3.22 to 13.55 mg per 100 cc, av. 7.17. Urine ammonia-N—12 determinations on 7 cows after parturition, range 4.54 to 13.44 mg per 100 cc, av. 9.75.

In general, there was a slight decrease in the CO_2-combining power of the blood plasma in late pregnancy. This was usually followed by a rise and then a decrease to the normal level during the first few days after calving. These results are not entirely in accord with those reported by Blatherwick[5] for five pregnant cows, but agree in general with some results published by Trautmann and Koch[11] for the alkali reserve of two pregnant goats. The data accumulated in this study for the serum calcium agree with the findings of Godden and Allcroft.[12] The values for total blood ketones reported here are somewhat lower than the results found by Christalon[13] for pregnant and nonpregnant cows, and steers. The normal range and the average for total urine ketones in this study were found to be much lower than the range and average reported by Little and Wright[14] and by Sjollema and Van Der Zande[15] for the urine of cows. The values reported here agree more closely with the data published by Carpenter[4] for the urine of steers.

*All results for blood and urine ketones are expressed as acetone.

Milk Fever

With the exception of two cases out of 34 that were diagnosed as either milk fever, atypical milk fever, complications plus milk fever, or "palsy after calving,"* 20 were classed as *typical milk fever,* nine as a combination of *milk fever with acetonemia,* and five as *typical acetonemia.* This classification was based on the calcium content of the blood and the concentration of the ketones in the pretreatment samples of blood and urine taken from the affected cows. The justification for such a classification is discussed by us elsewhere.[16]

Blood and urine analyses: CO_2 capacity of plasma—5 cases, range 54.5 to 64.5 vol. per cent, av. 60.54. Serum calcium—18 cases, range 2.37 to 7.50 mg per 100 cc, av. 4.09. Total blood ketones—17 cases, range 2.40 to 6.09 mg per 100 cc, av. 4.29. Total urine ketones—12 cases, range 4.48 to 15.78 mg per 100 cc, av. 8.14 Urine ammonia-N—7 cases, range 2.19 to 25.51 mg per 100 cc, av. 9.45.

The average reported here for the alkali reserve is slightly higher than the average of 58.8 vol. per cent found by Sjollema[17] for 31 cases of milk fever, but it is still 3.82 vol. per cent lower than the average of 64.32 obtained in this study for cows after calving. The range and the average for serum calcium are in agreement with the results reported by others for milk fever.[14, 18, 19]

Milk Fever with Acetonemia

Blood and urine analyses: CO_2 capacity of plasma—3 cases, range 46.6 to 65.5 vol. per cent, av. 55.63. Serum calcium—9 cases, range 2.85 to 10.75 mg per 100 cc, av. 4.83. Total blood ketones—8 cases, range 5.82 to 30.30 mg per 100 cc, av. 12.70. Total urine ketones—7 cases, range 8.36 to 267.85 mg per 100 cc, av. 66.23. Urine ammonia-N—6 cases, range 4.59 to 73.53 per 100 cc, av. 24.65.

Acetonemia

Blood and urine analyses: CO_2 capacity of plasma—1 case, 54.8 vol. per cent. Serum calcium—5 cases, range 7.60 to 9.50 mg per 100 cc, av. 8.83. Total blood ketones—5 cases, range 27.93 to 63.85 mg per 100 cc, av. 44.56. Total urine ketones—3 cases, range 35.10 to 1,209.67 mg per 100 cc, av. 578.25. Urine ammonia-N—3 cases, range 27.47 to 80.00 mg per 100 cc, av. 59.63.

*The samples of blood and urine from these two cases were kindly sent to one of us (Hayden) by Dr. J. T. Alston, of Tupelo, Miss. Dr. Alston diagnosed these cases as "palsy after calving."

These results agree, in the main, with those reported by Sjollema and Van Der Zande[15] and Sjollema,[20] for acetonemia in cows.

PNEUMONIA AND HEMORRHAGIC SEPTICEMIA

Blood and urine analyses: CO_2 capacity of plasma—15 cases (7 were pneumonia in calves), range 61.4 to 77.7 vol. per cent, av. 67.64. Serum calcium—9 cases, range 9.03 to 11.50 mg per 100 cc, av. 10.33. Total blood ketones—4 cases, range 1.71 to 3.33 mg per 100 cc, av. 2.41. Total urine ketones—1 case, 5.95 mg per 100 cc. Urine ammonia-N—1 case, 5.79 mg per 100 cc.

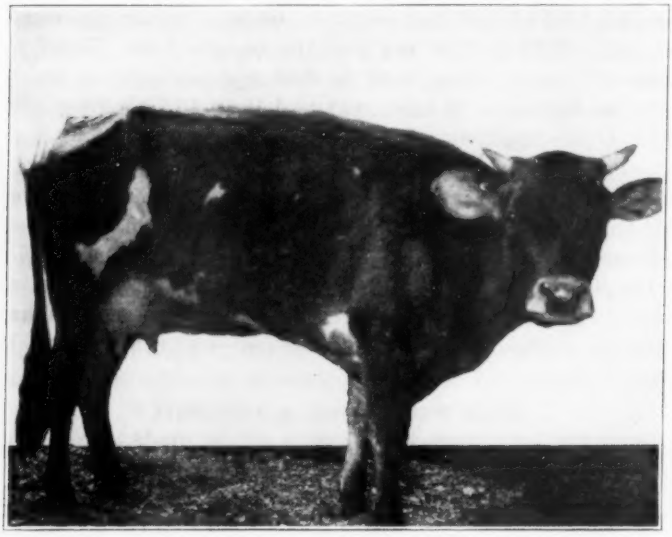

FIG. 1. Acetonemia in a two-year-old heifer. The attack occurred five weeks after calving.

OTHER DISEASES

This group includes three cases of septic metritis, three of dystocia, one of retained placenta, one of abortion, four of pyelonephritis, one of suspected intussusception, one of sweet-clover poisoning, one of gastroenteritis and peritonitis, four of an advanced stage of Johne's disease (one steer in this group), one of meningo-encephalitis, three of amaurosis (in calves), one of peritonitis, one of traumatic pericarditis and peritonitis, and two of fluorine poisoning.

Blood and urine analyses: CO_2 capacity of plasma—20 determinations, range 55.5 to 83.4 vol. per cent, av. 68.79. Serum cal-

cium—15 determinations, range 6.65* to 12.50 mg per 100 cc, av. 9.23. Total blood ketones—16 determinations, range 2.14 to 5.19 mg per 100 cc, av. 3.28. Total urine ketones—15 determinations, range 3.62 to 9.94 mg per 100 cc, av. 5.71. Urine ammonia-N—7 determinations, range 3.57 to 19.54 mg per 100 cc, av. 9.50.

The results for this miscellaneous group of diseases are practically all within normal range. It is significant that in this fairly representative group of abnormal conditions in cattle, the blood ketones do not show a concentration of more than 6 mg, and the urine ketones, not more than approximately 10 mg per 100 cc.

II. Results of the Studies with Ewes

The main objects in these studies were, first, to determine normal values for a reasonable number of ewes in late pregnancy and shortly after parturition; second, to study the blood and urine of ewes affected with pregnancy disease. A limited amount of data were accumulated also for several miscellaneous disorders in sheep. Unfortunately, only a few samples of urine were collected for normal ewes.

Late Pregnancy and One to Two Days After Lambing

Blood and urine analyses: CO_2 capacity of plasma—9 determinations for 9 pregnant ewes, range 59.8 to 72.0 vol. per cent, av. 64.20; 5 determinations for 5 ewes after parturition, range 58.6 to 70.0 vol. per cent, av. 64.30. Serum calcium—9 determinations for 9 pregnant ewes, range 8.07 to 10.45 mg per 100 cc, av. 8.71; 5 determinations for 5 ewes after parturition, range 9.50 to 10.45 mg per 100 cc, av. 9.97. Total blood ketones—9 determinations for 9 pregnant ewes, range 2.73 to 11.36 mg† per 100 cc, av. 4.12; 5 determinations for 5 ewes after parturition, range 2.68 to 3.26 mg per 100 cc, av. 2.92. Total urine ketones—2 determinations for 2 pregnant ewes, range 8.88 to 11.19 mg per 100 cc, av. 10.03. Urine ammonia-N—1 determination for 1 pregnant ewe, 4.46 mg per 100 cc.

These particular data show no characteristic changes in the blood in late pregnancy as compared with the blood of ewes after parturition. However, the data are too limited in number to have much significance from this standpoint. It is well to men-

*This value was found for the serum calcium of the blood of a cow that was destroyed; diagnosis, meningo-encephalitis with complications.

†This value is suggestive of moderate ketosis.

tion, also, that in only two instances were the same ewes studied both before and after lambing.

The results for urine ketones and ammonia nitrogen are hardly significant. Roderick and Harshfield[21] have reported, however, that only traces of acetone and ammonia are to be found in the urine of normal pregnant ewes. With one exception, all of the values for blood ketones were between 2.64 mg and 3.49 mg per 100 cc. If the value of 11.36 mg is excluded, the averages for total ketones in the blood of pregnant ewes and of ewes that had recently lambed approximate those for the blood of the cows in late pregnancy and during the first few days after calving.

The results for the CO_2 capacity of the plasma are higher than those reported by others.[21, 22] Additional data for the alkali reserve of normal sheep are desirable.

Pregnancy Disease

Blood and urine analyses. 1. *Pretreatment samples:* CO_2 capacity of plasma—9 cases, range 28.7 to 70.0 vol. per cent, av. 50.12. Serum calcium—9 cases, range 6.50 to 11.00 mg per 100 cc, av. 9.10. Total blood ketones—4 cases, range 36.23 to 48.56 mg per 100 cc, av. 42.17. Total urine ketones—4 cases, range 326.08 to 937.50 mg per 100 cc, av. 651.02. Urine ammonia-N—6 cases, range 16.66 to 114.90 mg per 100 cc, av. 81.01.

2. *Samples taken a few hours before or just at the time of death:* The values are averages. CO_2 capacity of plasma—6 cases, 54.15 vol. per cent. Serum calcium—6 cases, 10.35 mg per 100 cc. Blood ketones (preformed acetone and acetone from aceto-acetic acid)—3 cases, 2.82 mg per 100 cc. Total ketones—2 cases, 21.48 mg per 100 cc. Total urine ketones—2 cases, 83.18 mg per 100 cc. Urine ammonia-N—1 case, 44.85 mg per 100 cc.

3. *Samples taken at the time of recovery:* The values are averages. CO_2 capacity of plasma—2 cases, 56.65 vol. per cent. Serum calcium—2 cases, 10.21 mg per 100 cc. Total blood ketones—2 cases, 3.75 mg per 100 cc. Total urine ketones—1 case, 6.27 mg per 100 cc. Urine ammonia-N—1 case, 4.30 mg per 100 cc.

These data, though not extensive, lend support to the viewpoint first expressed by Dimock, Healy and Bullard[23] that pregnancy disease of ewes is characterized by acidosis. However, the results reported here, contrary to the findings of Dimock and his associates, tend to confirm the conclusion reached by Roderick and Harshfield[21] that a disturbance of the calcium metabolism

is not the predisposing cause of the acidosis. The data accumulated in this study substantiate, in many respects, the conclusions reached by Leslie[24] based on clinical and etiological studies of this disease in New England.

Miscellaneous Disorders in Sheep

Six cases of encephalitis and three cases characterized by some form of fatal digestive disturbance make up this group.

Blood and urine analyses (average values): CO_2 capacity of plasma—58.38 vol. per cent; serum calcium—9.78 mg per 100 cc; total blood ketones—4.21 mg per 100 cc.

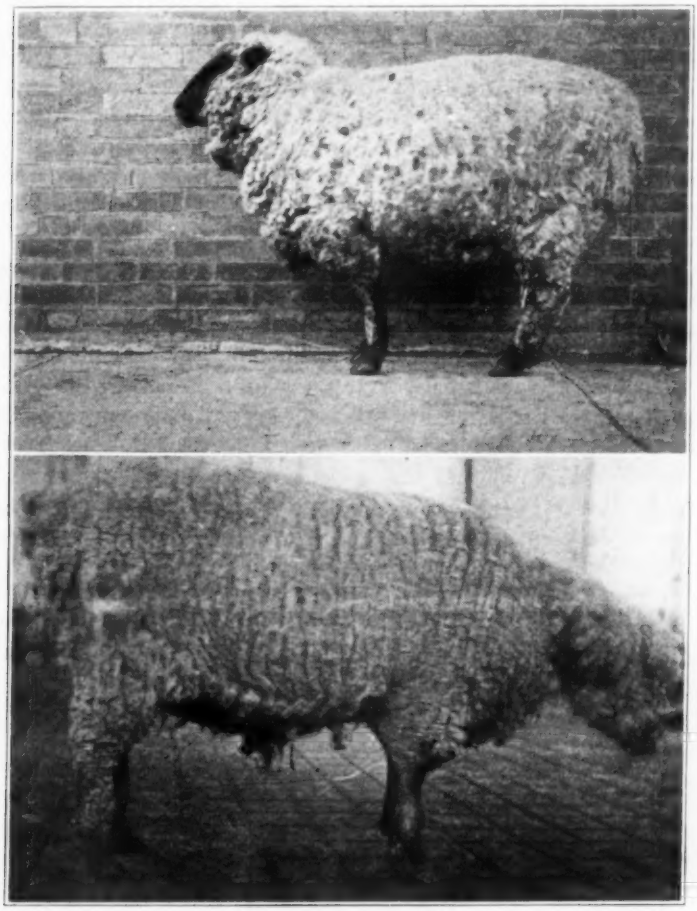

Fig. 2. Early stages of pregnancy disease of ewes.

An examination of the above data shows that these particular results are all within normal range.

DISCUSSION

The most striking and significant features of the results represented by the preceding data are the marked ketosis and the exceedingly high ketonuria associated with some of the pre- and post-parturient diseases of cows and ewes.

A comparison of the data for acetonemia of cows with the data for pregnancy disease of ewes shows that similar changes occur in the blood and urine of animals affected with these disorders.

Hupka[25] and Sjollema[20] have suggested that acetonemia of cows is possibly due to a disturbance of carbohydrate metabolism. Roderick and Harshfield[21] state that the physiological changes and symptoms in pregnancy disease are due to a faulty metabolism of carbohydrate. According to the viewpoint expressed by Peters and Van Slyke[26] as to the causes of ketosis in general, it is highly probable that there is no cause for the ketosis in these diseases other than some derangement in carbohydrate metabolism.

As there seems to be no evidence to indicate that the power to oxidize carbohydrate is impaired in acetonemia[20] and pregnancy disease,[16] one or the other of the first two conditions mentioned by Peters and Van Slyke[26]—(1) an insufficient exogenous supply of carbohydrate or carbohydrate-forming material in the diet for the amount of fat burned in the body or (2) deficient endogenous carbohydrate stores—must be responsible for the ketosis found in these cases. Whether the depletion of the carbohydrate in the body is due to the demand for glucose by the rapidly developing fetus or fetuses in the pregnant ewe (as suggested by Stander and Cadden[27] as a possible cause for the tendency toward ketosis in pregnant women) or to the requirement for carbohydrate in milk production in the cow,[20] the significant condition, in both disorders, would be the lack of a sufficient amount of glucose in the metabolic mixture for the amount of fat that is burned.

SUMMARY

An appraisal of the data reported for these studies on the acid-base balance in normal and diseased cows and ewes seems to warrant the following conclusions:

1. The alkali reserve is slightly lower in pregnancy (especially near the end) than during the first few days and perhaps weeks after parturition in cows.

2. The serum calcium and the blood ketones are slightly higher in pregnancy than during the first few days after parturition in cows.

3. There is a lowering of the alkali reserve and serum calcium in (a) milk fever, (b) milk fever with acetonemia, and in (c) acetonemia of cows. The calcium average is about one-half the normal average in the first two of these disturbances.

4. The ketones of the blood and urine show an increase, especially in those cases classed as a combination of milk fever with acetonemia and as acetonemia. In the latter disorder, the increase may be more than 20 times greater than the normal average for blood and over 150 times the normal average for urine (expressed as mg of acetone per 100 cc).

5. The average for urine ammonia-N shows a definite increase beyond the normal range in milk fever complicated with ketosis and in those cases classed as acetonemia.

6. The alkali reserve, serum calcium, blood and urine ketones, and urine ammonia-N are within the normal range in a surprisingly large number of abnormal conditions in cattle.

7. The data accumulated in these studies of several of the post-parturient diseases of cows suggest the existence of a close etiological relationship between milk fever and acetonemia.

8. A marked ketosis and an extremely high ketonuria are usually associated with pregnancy disease of ewes.

9. It is highly probable that the essential cause of the severe ketosis associated with acetonemia of milk cows and pregnancy disease of ewes is a disturbance in carbohydrate metabolism, due either to a lack of sufficient carbohydrate or carbohydrate forming material in the ration or to an insufficient supply of carbohydrate stored in the body.

METHODS

The CO_2 capacity of the plasma (alkali reserve) was determined by the method of Van Slyke and Cullen.[28] The colorimetric method of Behre and Benedict[29] was used for the determination of the blood and urine ketones. The color was developed by the aid of Kahlbaums' "Salicylaldehyd snyth." Serum calcium was determined by the Clark-Collip[30] modification of the Kramer-Tisdall method. Ammonia-N in the urine was determined by the permutit method of Folin and Bell.[31]

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New York Still Has a Place for the Horse

For the first time in a quarter of a century, New York City recently received a freight car of horseshoe nails. The horse, it appears, is coming back in the city as well as in the country, according to B. C. Forbes, well-known economist, in a syndicated article appearing in the Hearst chain of newspapers. Speculation arises, however, as to how these 2,323,800 nails—enough to shoe 72,618 horses—are going to be used. On first thought, it might look as if New York were going back to horse-cars. Or that New Yorkers expect to claim a share of the funds of the Agricultural Adjustment Administration, heretofore distributed only in farm areas. At any rate, facts are facts, and New York now has a carload of horseshoe nails available for those animals that are rapidly regaining a popularity that was eclipsed by the novelty of the motor car and the tractor.

Vice-President Carlos H. French, of the Fowler & Union Co., Buffalo, N. Y., points to the shipment as definite evidence of the return of the horse in industry. "For more than two years," he says, "a decided up-curve in the horseshoe nail business, coming from agricultural regions, proved to us that the farmers were on the way back to prosperity."

It is further pointed out that horses are now selling at an all-time high price. The national horse population has risen to above 17,000,000. This will do much to absorb the production from the millions of acres thrown into surplus crops since 1920 through the shrinkage of the horse population. New York City shows that it has again accepted the horse in the economic scheme of things.

SEROLOGICAL STUDY OF PIGEON PARATYPHOID IN EGYPT*

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During the summer of 1930, an epizoötic appeared among the pigeons of the Italian Hospital at Cairo. A number of sick and dead birds were brought to the Veterinary Pathological Laboratory for diagnosis, and from some of the cases we were able to isolate an organism of the Salmonella group. The object of this report is to give briefly the cultural and biochemical reactions of the organism isolated, to record some observations on its pathogenicity for some of the laboratory animals and, finally, to establish serologically its relationship to the other members of the Salmonella group.

ISOLATION OF THE ORIGINAL STRAIN

The bacteriological technic employed at autopsy was as follows: A piece of the spleen was removed aseptically and transferred to an agar slant. A large quantity of the heart-blood was transferred by pipette to a second agar slant. These were examined after incubation for 24 to 48 hours. If growth had occurred, microscopic examination was made and single colonies were transferred to tubes of plain bouillon. These were examined for motility after incubation for 18 hours and transfers were made to lead acetate agar and carbohydrate media in fermentation tubes.

In all cases examined, we were able to obtain from spleen and heart-blood an organism that showed cultural and biochemical characteristics of the paratyphoid members of the Salmonella group.

CULTURAL CHARACTERISTICS

Morphologically, the microörganism isolated was a short, Gram-negative, non-sporulating, actively motile bacillus. On ordinary agar, it produced flat non-mucoid colonies with regular borders. In broth, it produced a dense, even turbidity in 24 hours. The organism blackened lead acetate in peptone agar in 24 hours (hydrogen sulfid). Indol was not produced but there was a transient acidity in litmus milk, with a slow change to permanent alkalinity. Gelatin was not liquefied. Dextrose, maltose, mannite, galactose, levulose, xylose, sorbite and inosite were fer-

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mented, with the production of both acid and gas. There was no fermentation of lactose, sacchrose, dextrine or raffinose.

PATHOGENICITY

When freshly isolated, the organism was found to be pathogenic for pigeons, rabbits and guinea pigs when injected subcutaneously and sometimes when given *per os*. The course of the disease in experiment animals and the pathological manifestations encountered at autopsy varied considerably with the different species.

Guinea pigs: Subcutaneous injections of 0.5 cc of a 24-hour bouillon culture usually produced death in eight days. At autopsy, the spleen was found markedly enlarged, with its surface studded with foci varying in size from minute white points to large yellowish nodules. The liver often showed foci varying in number and size. The intestinal tract, particularly the small intestine, showed marked congestion. Cultures indicated the presence of the organism in the spleen, liver and blood-stream. They could rarely be demonstrated in films.

Rabbits: Subcutaneous injections of 0.5 cc of a 24-hour bouillon culture usually were fatal by the sixth day. At autopsy, the spleen was found markedly enlarged, but without focal changes. The intestinal tract showed marked congestion.

Pigeons: The same dose injected subcutaneously was fatal after five days, with slight enlargement of the spleen and marked congestion of the alimentary tract.

Serological studies: The relationship of the organism isolated to the other known members of the Salmonella group was studied first by direct agglutination. For this purpose, rabbits were immunized by intravenous injections of 18-hour bouillon cultures killed with 0.1 per cent formaldehyde. Each rabbit received four injections: 0.1 cc, 0.2 cc, 0.5 cc and 1 cc. Eight days after the fourth injection, each rabbit was bled from the ear vein and the serum tested for the presence of agglutinins.

The serum of the rabbit immunized against the pigeon paratyphoid organism agglutinated its homologous strain in a dilution up to 1:20,480. It agglutinated *Bacillus aertrycke* up to 1:10,240; *Bacillus paratyphosus* B up to 1:2,560, and *Bacillus paratyphosus* C up to 1:5,120 (table I).

The slight variation in the titre limits of this serum toward the pigeon paratyphoid and *B. aertrycke* is due perhaps to a difference in the proportions of the specific and non-specific factors contained in the antigens. The pigeon paratyphoid strain was recently isolated from the animal body and, for this reason, the non-specific factors are not expected to be present to an extent

such as is the case with the *B. aertrycke* strain that had been kept for years under laboratory conditions. Direct agglutination indicates that the epizoötic and the *B. aertrycke* strains are closely related.

It is desirable to make clear that the differential agglutination reactions referred to in this study depend solely on the agglutinating factors of the (H) or flocculating heat-labile factors.

For further study, absorption tests were conducted. For absorbing the agglutinins, a 20-hour living culture grown on the surface of agar was used. First, bouillon tubes were inoculated from the stock cultures and incubated for four to six hours. This growth was used to seed ordinary agar in pint Blake bottles. Two bottles were used for each strain. The growth in each bottle was suspended in a few cc of physiological salt solution. The cultures from the two bottles were mixed and further diluted with saline to 9.75 cc. To this was added 0.25 cc of the serum to be absorbed. The absorbing dose was allowed to act for about four hours at 37° C., and then transferred to the ice-chest and kept overnight. The next day, the mixture was centifuged until the supernatant fluid became clear. This clear, diluted, absorbed serum then was

TABLE I—*Agglutination with pigeon paratyphoid serum (produced from rabbit 1280).*

CULTURE	LIMIT OF AGGLUTINATION				
	UNABSORBED	ABSORBED WITH			
		PIGEON PARA.	AERTRYCKE	PARA. B	PARA. C
Pigeon paratyphoid..	1:20,480	1:320	1:320	1:20,480	1:10,240
Aertrycke.....	1:10,240	1:80	1:80	1:5,120	1:2,560
Paratyphoid B.....	1:2,560	0	0	0	0
Paratyphoid C.....	1:5,120	0	0	0	0

0=No agglutination at 1:80.

TABLE II—*Agglutination with aertrycke serum (produced from rabbit 2855).*

CULTURE	LIMIT OF AGGLUTINATION				
	UNABSORBED	ABSORBED WITH			
		PIGEON PARA.	AERTRYCKE	PARA. B	PARA. C
Pigeon paratyphoid...	1:5,120	1:80	1:80	1:5,120	1:5,120
Aertrycke.....	1:5,120	1:80	1:80	1:5,120	1:5,120
Paratyphoid B.....	1:2,580	0	0	0	0
Paratyphoid C.....	1:2,580	0	0	0	0

0=No agglutination at 1:80.

used for agglutination tests, with the results shown in tables I and II.

It is noted that the heavy absorption treatment of the pigeon paratyphoid and aertrycke serums, with paratyphoid B and paratyphoid C cultures, had no influence upon the titre limits of these serums for either the aertrycke or pigeon paratyphoid strains, while nearly all of the agglutinins for paratyphoid B and paratyphoid C were absorbed. But, on using the pigeon paratyphoid or the aertrycke culture for absorption, cross-absorption of agglutinins was observed. However, it is usually the case that in these cross-absorption experiments, a small residual titre always remains. Yet the results obtained render any positive differentiation of the epizoötic strain and the aertrycke strain out of the question. For all practical serological purposes, they are nearly identical. During the year 1931, we came in contact with two other separate epizoötics of the same kind, and Dr. M. Awad, of this Laboratory, was able to isolate the organism responsible for the epizoötics. These proved to be identical with the strains studied.

Something should be said concerning the rôle which this organism plays in outbreaks of food-poisoning. As far as can be determined from published reports, *B. aertrycke* has been isolated in typical outbreaks of food-poisoning more frequently than any other paratyphoid bacillus. But, as far as is known, no outbreaks of food-poisoning have been attributed to the consumption of pigeon meat, and it hardly would seem justifiable to discard altogether the possibility of this disease occurring in a country like Egypt where pigeon meat is consumed rather commonly. Cases may have occurred and passed unnoticed.

A Layman's Idea

A veterinarian of Melbourne, Australia, was tuberculin-testing a herd of cattle, when the owner picked up one of the bottles labeled "Koch's Old Tuberculin." "Oh," said he, "I see you use *old* tuberculin—I suppose you get it cheap."

Australian Veterinary Journal.

Oldest Salt Mine

The oldest salt mine in America is located at Avery Island, La. Pillars of salt in this mine are 60 feet high, and the salt is so pure and clear that printing can be read through a two-inch block of it.

THE EFFECTS OF FLUORINE ON RESPIRATION, BLOOD PRESSURE, COAGULATION, AND BLOOD CALCIUM AND PHOSPHORUS IN THE DOG*

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INTRODUCTION AND LITERATURE

The physiological effects of fluorine on the animal organism have been the subject of numerous investigations of the past. Most of these experiments have been confined to the following problems: 1. The distribution and function of fluorine in the animal body. 2. The relation between ingestion of small quantities of fluorides on food consumption, growth and reproduction. 3. The effect of fluorine on the development of teeth and bones. 4. The effect of fluorine on enzymatic changes pertaining to fat and carbohydrate metabolism. McClure[1] recently published a review of fluorine and its physiological effects, and there is appended to his article a comprehensive bibliography; so in view of this, only brief mention will be made of previous work relating to fluorine in general.

Considerable interest has been manifested in fluorine during the past few years. This is due to two reasons: First, fluorine is found in several phosphatic mineral supplements which are fed to farm animals; and, second, fluorine has been found in drinking waters in this and many other countries of the world. The interest of the authors in fluorine dates from the discovery by Ostrem, Nelson, Greenwood and Wilhelm[2] of fluorine in the drinking waters of communities in the state of Iowa, with the resulting production of mottled enamel. Recent work by McClure and Mitchell,[3] Tolle and Maynard,[4] Kick, Bethke and Edgington,[5] Hauck, Steenbock, Lowe and Halpin,[6] Phillips, Lamb, Hart and Bohstedt,[7] and Reed and Huffman[8] show that excessive fluorine present in phosphatic mineral supplements causes mottled enamel, inhibits growth, decreases food consumption and produces other harmful effects in farm animals.

Smith, Lantz and Smith,[9] Churchill,[10] McKay,[11] Ostrem, Nelson, Greenwood and Wilhelm,[2] and Dean[12] have produced considerable evidence that the presence of excessive fluorine (more

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than one or two parts per million) in the drinking water will cause mottled enamel in man. McCollum, Simmonds, Becker and Bunting[13] and Schulz and Lamb[14] observed some years ago that the feeding of sodium fluoride had a marked effect on metabolism and caused changes in the teeth of rats comparable to those we now recognize as characteristic of mottled teeth in human beings. Man and farm animals consume considerable fluorine in those areas where the water contains this element; and farm animals ingest additional fluorine if fed certain phosphatic minerals. Hence it seemed desirable to determine if fluorine produces other harmful effects or pathological changes in the body, aside from the mottled enamel of the teeth.

The work in this paper relates to the effect of fluorine on respiration, blood-pressure, hemoglobin, blood calcium, blood phosphorus, and clotting time of blood. Little has been done on these subjects. Sollmann[15] refers to the work of Tappeiner and states that in mammals sodium fluoride causes salivation, gastroenteritis, dyspnea, muscular weakness and tremors, epileptic convulsions, fall of arterial blood-pressure, and stoppage of respiration and heart. Schulz[16] likewise observed effects similar to Tappeiner. Schwyzer[17] states that fluorine has an effect on the formed elements of the blood, and that there is an irritation and inflammation of the bone-marrow. Gerschmann[18] reports that the injection of 30 mg of sodium fluoride per kilo body weight reduces blood calcium in 24 hours. McClure and Mitchell[3] have produced direct experimental evidence of an effect of ingested fluorine compounds on calcium metabolism. Stuber and Lang[19] observed a number of hemophilic patients with high amounts of fluorine in the blood, and they believed fluorine was the responsible factor. They cite considerable evidence that a high fluoride content of blood acompanies a long clotting period. Schwyzer,[17] however, observed a very high degree of coagulability of the blood of rabbits, dogs and pigeons which received fluorine. The coagulation of normal rabbit blood in an open vessel required six to eight minutes; whereas, the coagulation time of blood from rabbits receiving fluorine was four minutes in one instance and two and one-half minutes in another. Hauck, Steenbock, Low and Halpin[6] have reported a few analyses of the calcium and phosphorus content of the blood of chickens receiving fluorides. However, they admit that their data are insufficient to draw any definite conclusions. Phillips[20] summarized concisely the status of this problem when he stated:

> Very little work is reported dealing with the influence of fluorine upon blood composition.

Description of Animals

All of the experiments were performed on dogs. A total of seven dogs, five females and two males, were employed in studies on respiration and blood-pressure. The smallest of these seven dogs weighed six kilograms and the heaviest 26.8 kilograms. The exact age of the dogs was not known, although they were all mature animals. The remainder of the experiments were performed on 13 puppies. Nine of the puppies were born in this laboratory; the remainder were purchased when four weeks of age. The distribution of the puppies by sex was five males and eight females. The puppies were mongrels of about the same age and weight. All of the dogs were confined in cages composed of wooden frames, to which was fastened hardware cloth. Shavings were used as litter.

Anesthesia

Nembutal, sodium ethyl (1-methyl-butyl) barbiturate, dissolved in distilled water, was employed as the anesthetic. In general it was administered intraperitoneally at the rate of 30 mg per kilo of body weight. However, dogs 3 and 5 required 45 and 60 mg of this anesthetic respectively per kilo of body weight instead of the regular 30 mg. This may have been due to differences in the ages of the animals.

Methods and Apparatus

Blood-pressure records were taken from the central (heart) end of a ligated carotid artery with a mercury manometer writing on a long paper kymograph. Eight per cent sodium citrate solution was used to prevent formation of blood clots in the arterial cannulae and the tubes. The respiratory tracings were made with a Becker air tambour. The receiving end of the system consisted of a pneumograph tied around the chest. Time tracings were made in all of the experiments on respiration and blood-pressure. The animals were kept in a horizontal position, always on the back. Sodium fluoride was injected into the femoral vein of five of the dogs by means of a cannula. Dogs 5 and 6 received the fluoride orally. Newcomer's method involving the formation of acid hematin was employed in the hemoglobin determinations. The Brodie-Russell-Boggs coagulometer was used in studying the time of coagulation of blood. Calcium was determined in the blood plasma by the Clark and Collip modification of the Kramer-Tisdall method and phosphorus by the procedure of Youngberg. Sodium citrate (60 mg citrate per 10 cc blood) was employed as an anticoagulant in the calcium and phosphorus

determinations. The citrate was placed in test-tubes, cooled in ice, to which was added blood from the external saphenous vein; the mixture was centrifuged immediately, and the calcium and phosphorus determinations were made promptly.

Experimental

Table I summarizes the results obtained on respiration and blood-pressure; the data show that the intravenous injection of 1.5 to 5.3 mg of F as NaF per kilo of body weight produces a detectable effect on respiration. The average respirations were increased from 15 per minute to 22 per minute, by injecting from 1.5 to 5.3 mg of F as NaF per kilo of body weight. Furthermore, the data reveal that the intravenous injection of 16 to 31.7 mg of F as NaF per kilo of body weight causes a perceptible lowering of blood-pressure. The average blood-pressure was lowered from 170 mm Hg pressure to 135.6 mm Hg pressure, by increasing the concentration of F as NaF from 0 mg per kilo body weight to a level of 16 to 31.7 mg of F as NaF per kilo body weight. The above amounts of NaF were the initial doses. At intervals following the initial dose, larger amounts of NaF were injected into the femoral vein. Following increased doses, respirations increased to as high as 150 per minute, and blood-pressure fell to as low as 40 mm Hg. In all cases there was respiratory paralysis before stoppage of the heart. Two dogs received orally, per kilo of body weight, 20 times the amount of F as NaF ordinarily consumed daily by human beings in an endemic area (Ankeny, Iowa) without causing any noticeable effect on respiration and blood-pressure. The dogs received orally 4.5 mg of F as NaF per kilo of body weight, which was followed in a few minutes by 9 mg of F as NaF and then up to higher doses at intervals of a few minutes. When the oral dosage of F reached 22.6 mg per kilo of body weight, a detectable effect on respiration and blood-pressure resulted. At the higher levels vomiting and salivation occurred, which prevented accurate determinations. Autopsy of these dogs showed gastroenteritis and congestion of the kidneys. Dr. A. E. Merkel, of Ankeny (an endemic, mottled-enamel area), has made blood-pressure studies on 65 school children who received fluoride water and exhibited mottled enamel, and he has observed that there is no significant variation of blood-pressure from the normal. A typical blood-pressure and respiratory tracing is shown in figure 1.

Determinations of hemoglobin, coagulation time of blood, blood calcium, and blood phosphorus were made every two weeks for

TABLE I—*Effect of intravenous injection of fluorine as sodium fluoride on respiration and blood-pressure.*

DOG	RESPIRATIONS PER MINUTE BEFORE INJECTION OF F	MG F PER KILO BODY WEIGHT REQUIRED TO PRODUCE DETECTABLE CHANGE IN RESPIRATION	RESPIRATIONS PER MINUTE AFTER INJECTION OF F	BLOOD-PRESSURE IN MM HG BEFORE INJECTION OF F	MG F PER KILO BODY WEIGHT REQUIRED TO PRODUCE DETECTABLE CHANGE IN BLOOD-PRESSURE	BLOOD-PRESSURE IN MM HG AFTER INJECTION OF F	MG F PER KILO BODY WEIGHT WHICH CAUSED DEATH OF ANIMAL
1	*	....	....	145	31.7	120	92.6
2	12	5.3	16	150	20.4	114	47.5
3	†	....	....	200	23.8	164	32.5
4	12	1.5	20	180	22.7	120	30.4
7	22	2.0	32	175	16.0	160	31.9
Average	15.3	2.9	22.6	170	22.9	135.6	47.0

*Artificial respiration was used during the first part of the experiment.
†Artificial respiration was used throughout the experiment.

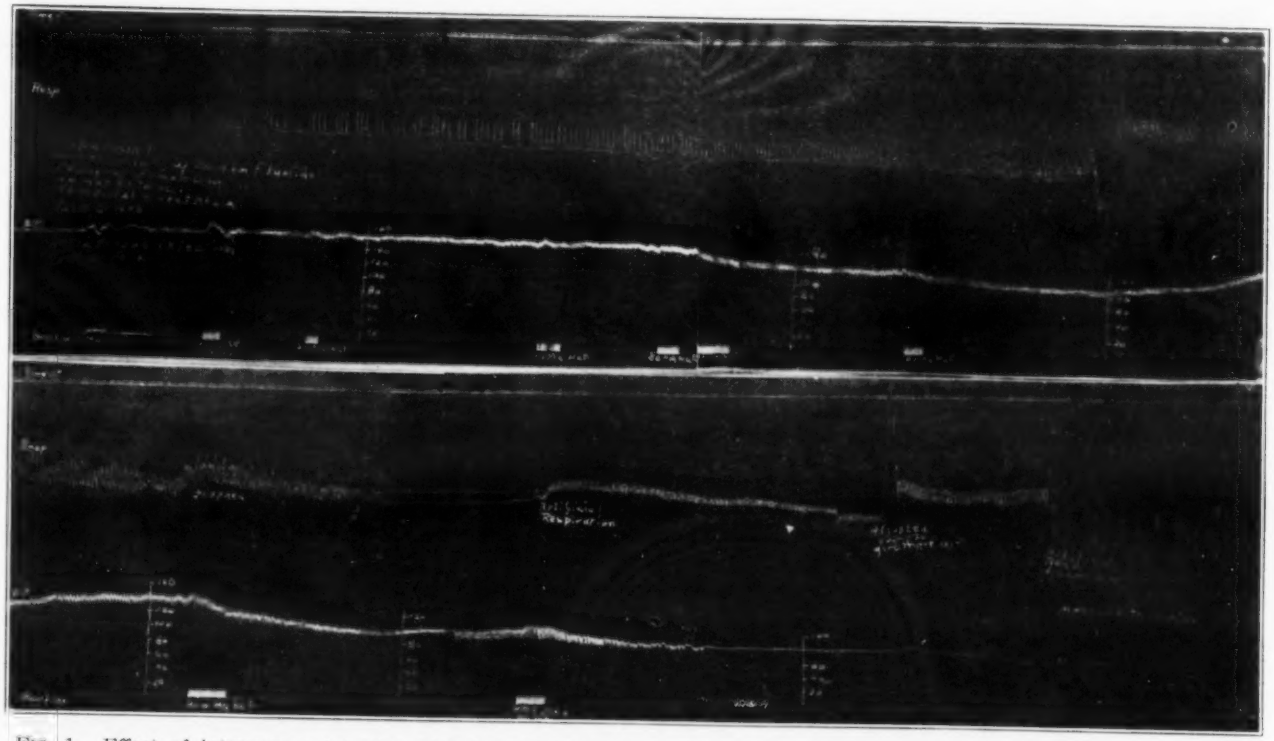

FIG. 1. Effect of intravenous injection of sodium fluoride on respiration and blood-pressure. (The lower portion of the figure is a continuation of the top portion.)

18 weeks. The analyses were made on 13 dogs, five to nine months of age. The puppies used in these experiments were originally divided into three lots: Lot I consisted of four puppies, three females and one male. Each dog in this lot received daily 180 cc of fresh whole milk and Purina dog chow and distilled water *ad lib.* Lot II consisted of four puppies, three females and one male. Each dog received daily 180 cc of fresh whole milk and Purina dog chow and distilled water *ad lib.* To the milk was added fluorine at the rate of 0.45 mg per kilo of body weight. This quantity of fluorine represents about the average amount of this element consumed by the average individual in the region (Ankeny) of the state of Iowa where mottled enamel is endemic. The fluorine was obtained by evaporating drinking water from this region to dryness and then weighing the requisite amount of ash to be added daily to the ration of the dog. Lot III consisted of five puppies, 3 females and 2 males. Each dog received 180 cc of fresh whole milk and Purina dog chow and distilled water *ad lib.* To the milk was added daily 2.26 mg fluorine as sodium fluoride per kilo of body weight.

The puppies were born from March 18 to 21. The mothers and puppies had access to the above three rations until July 14. At that time the basal ration was changed. The new ration, which replaced the Purina dog chow, consisted of cereals 60 per cent (ground corn 30 and ground hulled oats 30) and 40 per cent of the following mixture: cooked Ideal dog chow 23 per cent, wheat germ meal 5.0 per cent, skimmed milk powder 10 per cent, cod-liver oil 1.0 per cent, and sodium chloride (containing 0.1 gm NaI per 1,000 gm NaCl) 1.0 per cent. The mixture of corn and oats was moistened with distilled water and heated in an autoclave at 15 pounds pressure for one and one-half hours; it was then dried and mixed with the other constituents. The amount of fluorine in the two rations was kept constant. The Boruff and Abbott[21] modification of the Willard and Winter method was employed for determination of fluorine.

The results of hemoglobin, coagulation time of blood, blood calcium, and blood phosphorus are given in tables II and III. The data show no significant changes in hemoglobin, blood coagulation, blood calcium, or acid-soluble inorganic phosphorus at the levels of fluorine administered. It is therefore evident that animals in endemic areas consuming water containing fluorides would not exhibit any change in these four factors. It is evident from table III that as the puppies increased in age the blood phosphorus decreased in concentration.

TABLE II—*Effect of fluorine as sodium fluoride on hemoglobin and coagulation time of puppies' blood.*

Mg of F Added per Kilo Body Weight to Ration per Day	0.0*	0.45	0.90†	2.26	4.52‡	0.0*	0.45	0.90†	2.26	4.52‡
Puppies.....	4	2	2	3	2	4	2	2	3	2
Date	Hemoglobin in Grams per 100 cc					Coagulation Time of Blood in Seconds				
7–28–33	11.0	10.6	11.0	11.3	11.2	90	70	60	95	90
8–11–33	11.7	11.5	11.5	11.5	11.5	85	60	90	94	84
8–23–33	11.0	12.3	11.5	11.0	11.0	80	60	80	94	94
9– 6–33	11.5	12.0	11.5	11.0	11.3	70	60	80	85	94
10– 7–33	11.3	12.8	12.5	12.3	12.5	70	60	89	105	95
10–21–33	12.0	12.5	12.5	12.5	12.5	85	80	92	103	91
11–11–33	12.5	12.5	13.0	13.0	12.5	82	90	89	98	95
11–25–33	12.0	13.0	13.3	12.0	13.0	90	75	91	90	91
12– 9–33	12.6	13.0	13.0	12.0	13.0	96	90	92	96	90
12–23–33	13.0	13.5	12.8	13.0	13.3	92	80	90	94	90
Average.....	11.86	12.37	12.26	11.96	12.18	84.0	72.5	85.3	95.4	91.4

*Rations contained trace of fluorine.
†Received 0.45 mg F per kilo of body weight until 9-6-33.
‡Received 2.26 mg F per kilo of body weight until 9-6-33.

TABLE III—*Effect of fluorine as sodium fluoride on calcium and phosphorus content of the blood plasma of puppies.*

MG OF F ADDED PER KILO BODY WEIGHT TO RATION PER DAY	0.0*	0.45	0.90†	2.26	4.52‡	0.0*	0.45	0.90†	2.26	4.52‡
PUPPIES.....	4	2	2	3	2	4	2	2	3	2
DATE	CALCIUM IN MG PER 100 CC IN BLOOD PLASMA					PHOSPHORUS IN MG PER 100 CC IN BLOOD PLASMA				
7–28–33	11.8	11.4	10.5	10.8	10.6	6.3	6.0	5.8	6.2	6.0
8–11–33	11.8	10.9	10.8	11.4	10.8	6.3	5.8	5.1	6.2	5.0
8–23–33	11.8	11.5	11.7	11.6	11.8	5.9	6.5	6.3	5.5	6.0
9– 7–33	11.5	11.5	12.0	12.8	12.5	5.8	6.4	6.5	6.2	5.6
10– 7–33	12.5	12.3	12.5	12.8	12.5	5.8	5.8	5.0	4.8	5.0
10–21–33	12.5	12.5	12.5	11.7	12.5	5.5	5.0	5.3	5.0	4.8
11–11–33	12.2	12.5	12.5	12.5	12.5	4.9	4.8	4.8	4.5	4.7
11–25–33	12.5	12.8	12.5	12.8	12.5	5.5	5.2	5.3	5.0	5.3
12– 9–33	12.6	12.5	12.5	12.6	12.5	5.0	4.8	4.5	4.5	4.6
12–23–33	12.5	12.6	12.5	12.5	12.5	4.8	4.5	4.3	4.5	4.3
Average.....	12.17	12.05	12.0	12.15	12.07	5.58	5.48	5.29	5.24	5.13

*Rations contained trace of fluorine.
†Received 0.45 mg F per kilo of body weight until 9-6-33.
‡Received 2.26 mg F per kilo of body weight until 9-6-33.

It is recognized from work in this laboratory and elsewhere that fluoride ingestion causes damage to the teeth; and that consumption of fluoride-containing waters causes changes in the teeth of experiment animals such as the rat, resulting in production of the dystrophy generally called mottled enamel. It was believed well worth while to determine whether such waters—or fluoride consumption—would produce the same effects on a larger animal, such as the dog. This animal differs from the rat in that it has both deciduous and permanent teeth; whereas, the rat has only one set of teeth, which continue to grow, because of a persistent pulp, during the life of the animal.

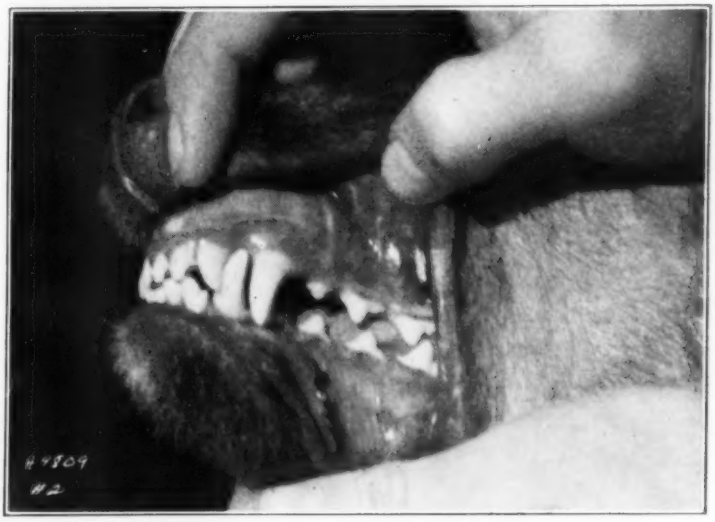

FIG. 2. Dog 2, the control or normal dog.

The puppies employed for determinations of hemoglobin, clotting time of blood, calcium, and phosphorus offered splendid material for this study, since these determinations were performed during part of the period of calcification and during the entire period of eruption. However, as stated above, no significant variations in blood calcium and phosphorus occurred. During this period of analysis, however, careful observations were made on the teeth of the dogs; and twice weekly the deciduous and permanent teeth were examined carefully.

As far as could be ascertained, no changes occurred in the enamel of the deciduous teeth of the dogs; but marked changes

occurred in the permanent teeth. Figures 2, 3 and 4 illustrate the teeth from three of the dogs: first, a normal dog; second, a dog which received drinking water from an endemic area, and third, another dog which received 2.26 mg of fluorine as sodium fluoride per kilo of body weight. These data show that dogs in areas where the concentration of fluorine in the water is sufficiently great will develop mottled enamel similar in appearance to that exhibited by man and the rat. However, it will be noted from these experiments that the deciduous teeth of the dogs were not affected. This is not necessarily true in man. Mottled deciduous teeth have been observed in human beings, but it is nevertheless true that mottled deciduous teeth are far less prevalent

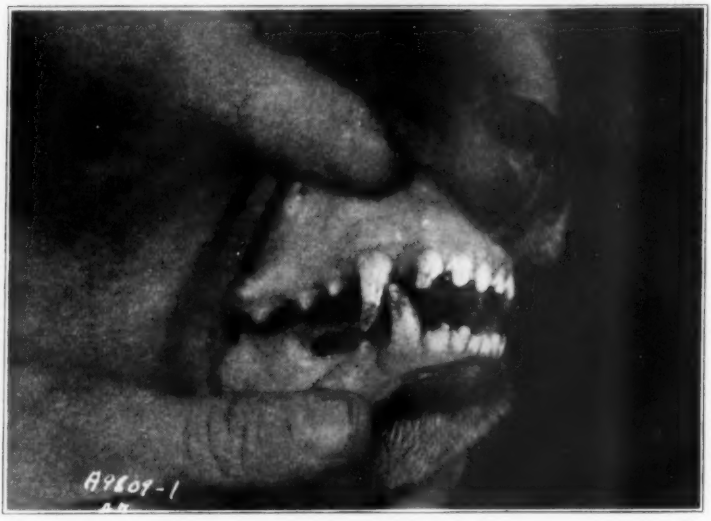

FIG. 3. Dog 7, which received 2.26 mg of F as NaF per kilo of body weight daily.

in man than mottled permanent teeth. It is possible that, if the biches had received the fluorine during pregnancy, dystrophy of the deciduous enamel may have resulted, since calcification of the deciduous and even the permanent teeth occurs, as is well known, during intrauterine life. It should be borne in mind, however, that fluorine, as sodium fluoride, and the fluoride water were fed to the bitches during lactation.

Møller and Gudjonsson[22] have reported on an examination of 78 workers engaged in the crushing and refining of cryolite. They found silicosis in 39 cases and in 30 cases a sclerotic affection of

the bones, ligaments, and muscular attachments, probably due to deposition of CaF_2. Radiographs of the bones revealed distinct increase in opaqueness; and the contours of the bones lacked in sharpness. The calcification changes were accompanied by stiffness of the limbs and impaired motility of the spinal column. The workers complained of rheumatism. They also reported that the workers suffered an acute affection of the stomach, due to slight corrosion of the mucous lining; complaints of nausea, lack of appetite, and vomiting were also noted. Pronounced anemia was exhibited by eleven workers who suffered osseous change. Blood calcium was normal.

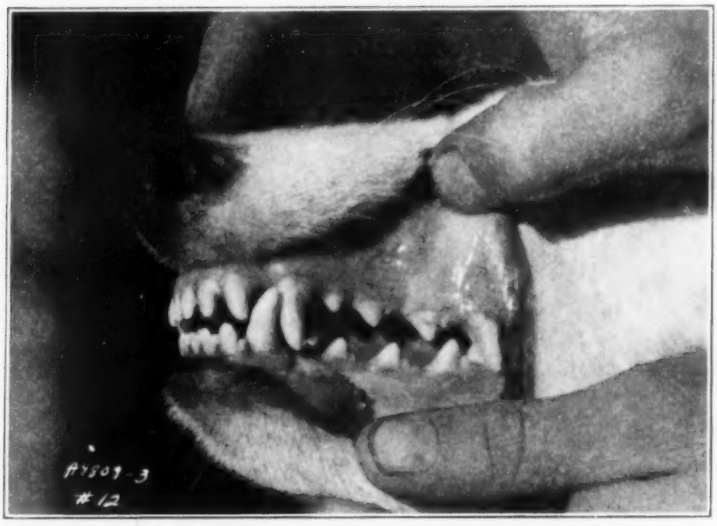

FIG. 4. Dog 12, which received 0.045 mg of F (from a drinking water containing F) per kilo of body weight daily.

Dr. G. H. Fowler, of the Department of Veterinary Surgery, was kind enough to make X-ray photographs of the head, front legs and hind legs of eight puppies used in the blood studies. These puppies received orally from 0.45 mg of fluorine to 4.52 mg of fluorine as sodium fluoride per kilo of body weight daily. Figure 5 shows an X-ray photograph of a normal radius and ulna and the radius and ulna from the puppy receiving 4.52 mg of fluorine as sodium fluoride per kilo of body weight. It is evident that this amount of fluorine gives rise to no changes detectable by X-ray; and none of the puppies examined revealed any characteristic changes of the bones by the X-ray method.

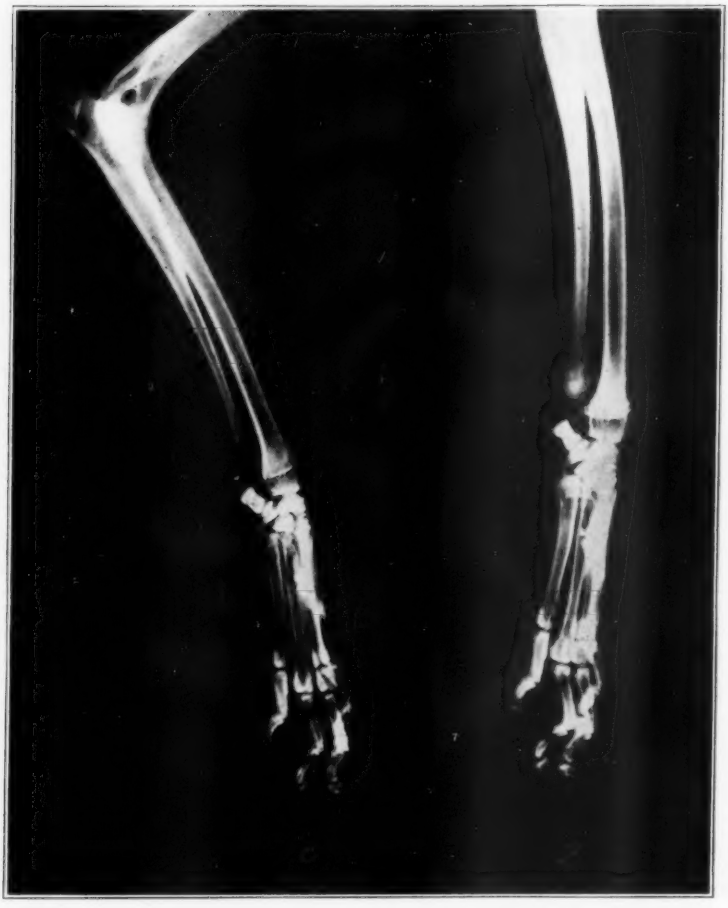

FIG. 5. X-ray photographs of fore legs of control dog and dog receiving 4.52 mg of fluorine as sodium fluoride daily per kilo of body weight. No. 8 is from the animal which received fluorine and No. 2 is from the control.

SUMMARY

1. Fluorine as sodium fluoride, when injected intravenously at levels from 1.5 to 5.3 mg per kilo of body weight, causes a detectable effect on respiration in dogs.

2. Intravenous injection of 16 to 31.7 mg of fluorine as sodium fluoride per kilo of body weight in dogs causes a detectable lowering of blood-pressure.

3. Oral administration of 0.45 mg to 4.52 of fluorine as sodium fluoride per kilo of body weight caused no effect on total calcium, acid-soluble inorganic phosphorus, hemoglobin, or coagulation time of blood.

4. Dogs developed mottled enamel of the permanent teeth when given orally fluorides or water containing fluorides.

5. Radiographs show no changes in the bones of dogs receiving as much as 4.52 mg of fluorine as sodium fluoride orally per kilo of body weight.

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Relief Meat in Toledo Vindicated

The relief meat distributed in Toledo, Ohio, and vicinity, which was the cause of much newspaper comment recently when several children in two of the city schools were reported ill after eating the meat, has received a clean bill of health, according to a report from the U. S. Bureau of Animal Industry. Not the meat, but intolerable sanitary conditions in the homes from which the children came were responsible for the digestive disturbances, according to the experts sent by the Department of Agriculture to study the situation at first hand, and to send in samples of the meat for laboratory examination.

Several food specialists and a number of trained meat inspectors, including a specialist in canned meats, made the tests, following a complaint by a representative of the Toledo Department of Health, who questioned the wholesomeness of the meat being distributed in the city because of a noticeable number of digestive disturbances among children in two of the city schools. The reports, which showed no evidence of spoiled meat, were based upon physical examination of large numbers of cans of the meat from the same lot that had been distributed and upon laboratory tests of many sample cans. Other bacteriological laboratories besides those of the government also studied the cases, and their findings were in accord with those reported by the Department of Agriculture.

Investigation revealed that usually only one or two persons out of several in a family had been affected, although in most cases others in the family had eaten of the meat. Few of the sick children were attended by physicians.

Separate tests were made by the Food and Drug Administration laboratory and the Meat Inspection Laboratories, Washington, D. C.

Bulletin on Rabbit Production

Rabbit raising as a commercial enterprise and as a minor farm industry is the subject of a dissertation by Frank G. Ashbrook and Charles E. Kellogg, issued as Farmers' Bulletin 1730-F. The bulletin is entitled "Rabbit Production," and sets forth the essentials of rabbit raising and explains how to apply them.

CROTALARIA SPECTABILIS ROTH SEED POISONING IN SWINE*

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In the fall of 1933, our attention was called to a condition which caused the loss of nine animals, over a period of three weeks, in one herd of hogs. The lesions presented on autopsy were strikingly similar in each animal but a definite diagnosis of the condition could not be reached at the time of the losses. The outstanding lesions found in these animals were a severe anemia, accumulations of fluid in the abdominal and thoracic cavities, a firm, indurated liver, ecchymoses of the endocardium, and gastritis often with subsequent hemorrhage.

Thomas[1] reported *Crotalaria spectabilis* Roth seed to be poisonous to chickens. More recently Emmel,[2] making further studies on this condition, found that a severe anemia may be induced in birds affected with the chronic type of *C. spectabilis* poisoning. Since the hogs in question had been kept in a field which contained a voluntary stand of *C. spectabilis*, this plant was suspected immediately as being responsible for the losses.

No reference could be found in the literature relative to crotolariosis in swine. Species of Crotalaria other than *C. spectabilis* have been found toxic for certain farm animals. Stalker[3] found *C. sagittalis* L. to be poisonous for horses and cows. Theiler[4] reported *C. burkeana* Benth. as inducing laminitis in cattle and *C. dura* Woods and Evans as being toxic to equines. Steyn and de Koch[5] reported *C. dura* to be poisonous for sheep. *C. globifera* was found poisonous for horses by Watt and Breyer-Brandwijk.[6] White[7] reported *C. striata* D. C. as being toxic for goats. *C. juncea* L. was reported by the Gwebi Experiment Farm[8] to be toxic for sheep.

Studies were undertaken to determine the toxicity of *C. spectabilis* seed for swine and to study the pathology of the condition produced.

EXPERIMENTAL DATA

EXPERIMENT I

In a preliminary experiment, a one-half ounce capsule of whole *C. spectabilis* seed was administered orally to each of a group of five 100-pound Poland China hogs daily for a period of ten days.

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These same hogs (1 to 5, inclusive) were then given orally two grams of the ground seed of *C. spectabilis* daily. The ground seeds were mixed with molasses and rolled into pellets which were placed over the root of the tongue with a pair of dressing forceps. Seven days after the first administration of ground seed, the hogs presented an unthrifty appearance. Thereafter this condition became more marked. One animal was killed on the tenth, another on the 12th, and a third one on the 14th day after exposure, while the remaining two were killed on the 18th day. (See table I.)

Gross lesions: Macroscopically numerous ecchymoses were found in the myocardium, more frequently in the right ventricle than in the left. Occasionally a few petechiae were observed on the surface of the auricles. The heart was usually soft and flaccid.

The cut surface of the kidney frequently showed definite, circumscribed hemorrhages in the medulla.

Passive congestion was indicated in the liver by its distinct purple color. Often the surface of the liver presented a rather mottled appearance, caused by the presence of numerous light brown, irregular foci ranging in size from a pinhead to an inch in diameter. The cut surface bled rather freely. The gall-bladder was occasionally thickened and gray in color.

The fundic and occasionally the cardiac portion of the mucosa of the stomach contained a large congested area, often six inches in diameter. The mucosa in this area was irregularly necrosed. The stomach contents in immediate contact with this area were stained with blood. The submucosa in several instances was edematous, reaching a thickness of one-half inch in one case.

The lungs were irregularly mottled with small, deep, pink areas.

Histopathology. Numerous areas of necrosis were present in the lymph-nodes, usually more marked near the capsule of the organ. There were also numerous foci of proliferation of connective tissue near the capsule, these areas extending irregularly towards the medulla. Numerous areas of congestion were often encountered. These lesions were always most extensive in the mesenteric lymph-nodes.

The spleen showed numerous small foci of proliferation of connective tissue and a few small foci of necrosis. Much congestion was in evidence throughout the organ, being particularly extensive in the malpighian corpuscles.

Cloudy swelling was present in the pancreatic cells as well as the cells of the isles of Langerhans.

The cardiac muscle was in a general state of cloudy swelling. The ecchymoses of the endocardium, a characteristic lesion of *C. spectabilis* poisoning in pigs, were diffuse subendocardial hemorrhages. Occasionally a small hemorrhage occurred in the myocardium.

The affected portion of the mucosa of the stomach showed considerable necrosis of the surface epithelium. This necrosis often extended slightly into the interglandular tissue, at which point a slight proliferation of connective tissue was in evidence. Considerable congestion was usually present in the mucosa. Occasionally there were small interglandular foci of necrosis. The glandular epithelium showed varying degrees of cloudy swelling. In many instances foci of histiocytes occurred at the base of the mucosa. The submucosa was invariably extremely congested and showed much edema; occasionally a hemorrhage occurred. The muscularis mucosa and the muscle coat of the stomach showed cloudy swelling.

The glomeruli of the kidneys usually showed extensive congestion; many were enlarged and edematous while a few showed evidence of degeneration. A general, well-advanced, cloudy swelling was present in the tubular epithelium of the cortex; much cellular débris was present in the lumen and occasional tubules and groups of tubules were practically denuded of epithelium. A few small intertubular foci of proliferation of connective tissue and of histiocytes occurred. The tubular epithelium of the medulla was in a state of cloudy swelling. Numerous large areas of congestion were present. There was an occasional small focus of proliferation of connective tissue in the intertubular tissue.

Passive congestion occurred extensively in the liver lobules. The congestion was confined principally to the area immediately surrounding the central vein. Occasionally, however, practically the entire lobule was affected. While there was no pigmentation of the hepatic cells in this area, the hepatic cells often were undergoing necrosis. In general, the hepatic cells were in a well-advanced state of cloudy swelling. In one animal, however, the hepatic cells also showed slight fatty degeneration. The degeneration of the hepatic cells often seemed to be more severe immediately under the capsule of the organ. The interlobular tissue was slightly edematous. Cloudy swelling occurred in the epithelium of the bile-ducts.

TABLE I—*Total and differential blood-counts of experiment animals affected with Crotalaria spectabilis Roth seed poisoning.*

PIG	EXPOSED	AUTOPSY	GROUND SEED FED DAILY (GM.)	ERYTHROCYTES	LEUCOCYTES	HEMOGLOBIN (SAHLI)	LYMPHOCYTES	POLYMORPHONUCLEARS	EOSINOPHILES	BASOPHILES	MONOCYTES	ERYTHROBLASTS PER 100 W. B. C.	POLYCHROMOPHILIA
1	11–27	12–6	2.0	4,980,000	17,200	63	29	63	4	1	3	21	.....
2	11–27	12–9	2.0	5,000,000	14,500	70	49	31	6	2	12	57	++
3	11–27	12–11	2.0	7,180,000	19,600	87	42	45	2	3	8	58	++
4	11–27	12–15	2.0	5,240,000	16,500	69	53	37	8	1	1	11	.....
5	11–27	12–15	2.0	6,870,000	14,500	72	39	46	12	1	2	10	.....
50	11–28	1–25	0.5	6,930,000	33,500	85	34	59	2	2	3	.....	.....
23	11–28	1–25	0.5	7,210,000	14,000	87	43	48	7	0	2	.....	++
43	11–28	1–25	0.5	8,590,000	19,400	90	60	36	1	2	1	.....	.....
14	11–28	1–25	0.5	8,020,000	10,500	88	39	53	6	0	2	.....	+
15	11–12	1–12	0.5	7,010,000	24,000	87	41	50	5	0	4	.....	.....
19	11–12	1–19	0.5	4,680,000	20,000	53	13	85	0	0	2	.....	.....
		1–18		7,950,000	21,000	83	59	32	5	0	4	.....	.....
				8,080,000	13,500	85	50	29	13	4	4	.....	+
				7,730,000	23,500	82	46	45	5	2	2	.....	.....
				8,070,000	20,500	81	12	88	0	0	0	.....	.....
				7,050,000	24,000	83	53	34	10	1	2	.....	.....
				6,000,000	14,500	83	54	33	11	1	1	.....	.....

TABLE I—*Total and differential blood-counts of experiment animals affected with Crotalaria spectabilis Roth seed poisoning—Concluded.*

PIG	EXPOSED	AUTOPSY	GROUND SEED FED DAILY (GM.)	ERYTHROCYTES	LEUCOCYTES	HEMOGLOBIN (SAHLI)	LYMPHOCYTES	POLYMORPHONUCLEARS	EOSINOPHILES	BASOPHILES	MONOCYTES	ERYTHROBLASTS PER 100 W. B. C.	POLYCHROMOPHILIA
58	11–28		0.5	7,070,000	21,500	80	49	46	3	0	2	.....	.....
		3–21	57 days	7,600,000	17,000	79	54	40	2	1	3	.....	.....
62	11–28		0.5	8,420,000	19,000	81	54	40	3	1	2	.....	.....
		3–21	57 days	7,550,000	17,500	81	47	46	4	0	3	.....	.....
17	11–28		0.5	5,320,000	13,500	78	36	58	3	1	2	.....	.....
		3–21	57 days	7,840,000	14,500	81	45	48	6	0	1	.....	.....
10	11–12		0.5	7,620,000	26,500	80	56	37	3	2	2	.....	.....
		3–21	57 days	7,540,000	20,000	81	49	42	5	2	2	.....	.....
72	Control		.....	5,880,000	14,000	80	42	52	3	1	2	.....	.....
		4–5		7,260,000	15,500	82	48	40	6	2	4	.....	.....
13	Control			7,250,000	21,500	81	60	36	1	1	2	.....	.....
		4–5		7,520,000	18,000	80	46	42	5	2	5	.....	.....
40	Control		.....	6,780,000	14,500	82	50	42	4	2	2	.....	.....
		1–5		7,460,000	13,500	85	58	35	3	1	3	.....	.....
63	Control		.....	8,120,000	16,500	89	61	35	2	0	2	.....	.....
		1–5		7,760,000	14,500	88	53	38	4	2	3	.....	.....
			Natural case after treatment*	2,290,000	13,000	54	55	36	5	0	4	3	.....
				6,760,000	16,000	86	70	27	2	0	1	.....	.....

*Treatment started December 19, and observations made on December 27, eight days later.

The epithelium of the gall-bladder showed cloudy swelling. In those cases in which the wall of the gall-bladder was thickened, the increased thickness was due to edema of the subserous and submucous tissues of the organ.

Irregular congestion and edema caused a thickening of the alveolar walls of the lungs. There were a few foci of histiocytes in the alveolar walls and in the peribronchiolar tissue.

The glandular epithelium of the thyroid gland was in a state of well-advanced cloudy swelling. Considerable débris filled the lumen of the glands. A few small interglandular foci of proliferation of connective tissue occurred.

EXPERIMENT II

Since chronic *C. spectabilis* seed poisoning has been observed in the chicken, one-half gram of ground seed daily was administered orally to each of a second group of ten hogs in the same manner as previously described, in an attempt to induce chronic cases of poisoning. Two animals (72 and 13) intended for controls were kept in the same pen, which had a concrete floor.

It is interesting to note that all of the pigs of this group to which ground seeds were administered began to lose their hair after 21 days. The pigmented hair was almost entirely shed in each instance before the white hair began to be shed. The haircoat of the control pigs remained normal. At this time the pigs fed ground seeds presented an unthrifty appearance, which gradually became more pronounced. An animal was killed on the 44th, another on the 50th, and a third on the 51st day after the first administration, while three were killed on the 56th day.

Gross lesions: The gross and microscopic pathology presented by these six animals was fundamentally the same in character as has been described previously. However, the intensity of the lesions was not so great. There was also slightly more proliferation of connective tissue in areas where this condition was found than in the animals of which the pathology has been described.

Histopathology: The lesions shown by this group of animals were fundamentally the same in character as those previously described. They were, however, less extensive.

EXPERIMENT IIa

The remaining four hogs of this group were placed in a dry lot, where they were held for 57 days after the last administration of ground seeds. During this time their general appearance

did not improve. At the conclusion of this period autopsies were held.

Gross lesions: The gross lesions found in these four animals were similar to those previously described, with the exception of those found in the liver and lymph-nodes. The liver was in a state of atrophic cirrhosis; the lymph-nodes, particularly the mesenteric, were almost white in color and very fibrous.

Histopathology: The outstanding features of the histopathology of these four pigs were the extensive proliferation of connective tissue in the liver and lymph-nodes, the persistence of general cloudy swelling throughout the visceral organs, and the diminution of lymphoid cells in the lymph-nodes and splenic cells in the spleen.

The lymph-nodes still showed scattered areas of congestion. Extensive areas of adult connective tissue were present particularly at the periphery of this organ and often extended irregularly into the medulla. Many irregular areas, especially in close proximity to the trabeculae, were devoid of lymphoid cells, only the reticulum of the organ remaining. The mesenteric lymph-nodes showed these changes more extensively. The hemolymph-nodes were least affected.

Numerous small areas of congestion appeared in the spleen, more especially in the malpighian corpuscles. A few small hemorrhages and irregular areas of adult connective tissue appeared in the splenic pulp more frequently near the capsule. Many large areas in the spleen were practically devoid of splenic cells with only the reticulum of the organ remaining. There were a few intracapsular hemorrhages.

The pancreatic cells, as well as the cells comprising the isles of Langerhans, were in a general state of cloudy swelling. In one animal the pancreatic cells showed slight fatty degeneration.

In the cardiac muscle cloudy swelling persisted, although only to a slight degree, the cells appearing in some instances to be atrophic.

The mucosa of the affected area of the fundic portion of the stomach showed necrosis of the surface epithelium. There was a slight active proliferation of connective tissue beneath. The glandular epithelium was in a state of cloudy swelling, this condition being more pronounced in the deeper glands. Occasionally the submucosa showed congestion. Cloudy swelling persisted to a slight extent in the muscular wall.

Many of the glomeruli of the kidneys were somewhat enlarged and usually edematous, others were often congested, while a few

showed evidences of degeneration. The tubular epithelium of the cortex was in a well-advanced state of cloudy swelling. Much débris was present in the lumen of the tubules. Occasionally a tubule was practically denuded of epithelium. In most instances, however, the epithelial cells had lost much of their cytoplasm. Pyknosis rarely was observed. The tubular epithelium in the medulla was in a state of cloudy swelling. Numerous small areas of congestion appeared in this portion of the kidneys.

The cytoplasm of the hepatic cells was extremely and rather coarsely granular, although the cells themselves and cords of liver cells were atrophic. There were many areas surrounding the central vein in which only the reticulum of the organ remained. In other instances, where the cells surrounding the central vein were still intact, slight deposition of pigment and passive congestion occurred. The capsule of the liver was irregularly thickened and often encroached upon adjacent lobules. The interlobular connective tissue, while not so abundant as would be suspected from the tough character of the liver, on gross examination was only slightly thickened. The greatest proliferation of connective tissue had usually taken place in the vicinity of the portal vessels. Slight edema often occurred in the periportal tissue. Irregularities in the interlobular connective tissue often extended into adjacent lobules of the liver. Occasionally, small, loosely arranged foci of histiocytes were observed in the interlobular tissue.

The submucosa of the gall-bladder showed considerable edema.

Few, scattered, but no extensive areas of congestion occurred in the alveolar walls of the lungs. Occasional small foci of histiocytes were present in the alveolar walls and peribronchiolar tissue.

Controls: Control animals 72 and 13 were kept in the same pen as the animals in experiment II. This pen had a concrete floor. Upon autopsy these animals showed lesions indicative of *C. spectabilis* poisoning. However, these lesions were not intensive. Two other animals (40 and 63) were kept in a pen to themselves. These animals failed to show lesions of *C. spectabilis* poisoning. This would seem to indicate that animals 72 and 13 came in contact with sufficient of the toxic principle of *C. spectabilis* in the feces or urine of other animals to induce lesions of poisoning.

NATURAL CASES

Our experimental studies have led to the diagnosis of *C. spectabilis* poisoning as being the cause of death in the nine cases in

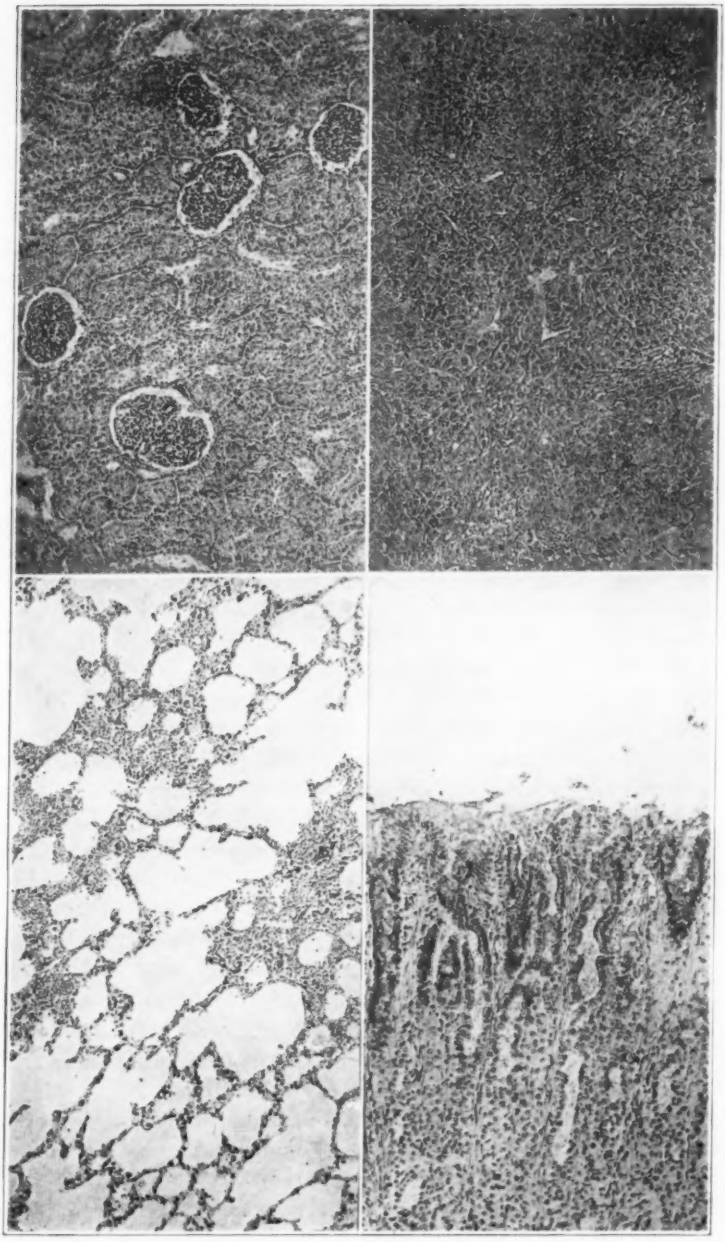

FIG. 1 (upper left). Photomicrograph of kidney (pig 43) showing enlargement of glomeruli, cloudy swelling and necrosis of the tubular epithelium (ca x 240).

FIG. 2 (upper right). Photomicrograph of liver (pig 15). Note passive congestion and cloudy swelling of hepatic cells (ca x 240).

FIG. 3 (lower left). Photomicrograph of lung (pig 50). Alveolar walls irregularly thickened by passive congestion (ca x 240).

FIG. 4 (lower right). Photomicrograph of mucosa of stomach (pig 15) showing necrosis of surface epithelium (ca x 240).

swine mentioned in the first part of this paper. All of these cases occurred in the same herd of hogs. The history of these natural cases of *C. spectabilis* poisoning is as follows: Approximately 25 sows of various breeds had been given access, during the month of July, to an acreage on which there was an extensive stand of *C. spectabilis* in a heavy growth of corn. The sows farrowed in this field and remained until the pigs were weaned. At this time both pigs and sows were removed to another pasture. However, the sows were placed back in the field containing *C. spectabilis* about the middle of October. They remained here until the first of December.

The first case of *C. spectabilis* poisoning was observed in two of the young pigs October 15. The presence of a severe anemia characterized the cases. Other lesions, previously described as characterizing *C. spectabilis* poisoning in our experiment animals, were found. Seven cases of natural poisoning occurred in the sows, the first to be noted occurring December 19, or almost three weeks after the animals had been removed from the field containing *C. spectabilis*. Autopsies were held on all of these animals, but due to the time that had elapsed after death no histopathological study was made of the tissues.

On December 22, two of the young pigs were killed and although they showed no clinical evidence of being affected, the viscera presented lesions similar to those that have been studied in animals experimentally exposed to *C. spectabilis* seed.

The outstanding lesions shown by these natural cases of *C. spectabilis* poisoning were anemia, accumulations of fluid in the abdominal and thoracic cavities, ecchymoses in the endocardium, particularly the right ventricle, and the presence of extensive gastritis. The gastritis was so severe in three cases that the animals died from gastric hemorrhage. Considerable variation occurred in the extent to which edema developed. In some cases edema was not evident macroscopically. In one animal extensive ascites, hydrothorax, and serous pericarditis were observed; the lungs in this instance were markedly edematous.

One sow, a purebred Berkshire, affected with *C. spectabilis* poisoning, was observed clinically. Temperature was normal, respirations and pulse slightly accelerated. The animal showed a general indisposition. The hemoglobin was 54 (Sahli). A blood count showed the erythrocytes to number 2,290,000 per cmm. After being observed for three days, during which the animal gradually became weaker, daily doses of 10 mg of copper sulfate and 10 mg of ferric ammonium citrate were administered. The

first three doses were given as a drench, after which the remedy was placed in the swill. The day following the first dose, improvement was noted, particularly by the fact that the animal ate voluntarily for the first time in three days. Recovery was extremely rapid, as noted by the blood count and hemoglobin reading taken on the eighth day after treatment was commenced. The erythrocytes at this time numbered 6,760,000 per cmm, while the hemoglobin reading was 86 (Sahli).

Discussion

Four of the experiment animals showed evidence of marked anemia, as indicated by a significant decrease in the number of erythrocytes and a similar decrease in the hemoglobin reading. Three of these animals received 2-gram daily doses of the ground seed of *C. spectabilis* over a period of nine to 18 days; the fourth received 0.5-gram daily doses over a period of 57 days.

These experiments do not seem to settle conclusively whether *C. spectabilis* poisoning in hogs is a subacute or a chronic condition. Much probably depends on the daily intake of the seed on the part of the animal. It is interesting to note that the first case of poisoning in sows was not observed until three weeks after the animals had been removed from the pasture in which they had access to *C. spectabilis*. The animals in the herd in which the natural cases occurred were not under close observation and, therefore, it is not known how long before death clinical symptoms may be observed. Evidence would indicate that this period may be of short duration.

As the first two pigs in which *C. spectabilis* poisoning was observed under natural conditions died October 15, it would seem that they were poisoned by the plant instead of the seed, as in the case of the experiment animals. This is particularly true since Neal, Ahmann and Rusoff[9] have isolated an alkaloid from the stems, leaves and seeds of *C. spectabilis* which is exceedingly toxic.

Neal *et al* have shown also that the principal physiological action of this alkaloid is that it reduces blood pressure. This activity on the part of the alkaloid is no doubt responsible for the fact that passive congestion and edema are so prevalent in most of the visceral organs. It may also explain why the animals fed 0.5-gram daily doses lost their hair-coat while the hair-coat of control animals in the same pen remained normal. Lack of nourishment may also explain the small foci of necrosis in the spleen, lymph-nodes and liver of the experiment animals.

Differential blood cell counts indicate that the number of lymphocytes is often greatly reduced. The reduction in lymphocytes is compensated for by an increase in polymorphonuclears. In the subacute cases of experimental poisoning, erythroblasts, 10 to 58 per 100 leucocytes, were found in the blood smears, the highest number being found in animal 3, which still showed an erythrocyte count of 7,180,000 per cmm and a hemoglobin reading (Sahli) of 87 at date of autopsy. Five animals showed a polychromophilia.

These studies are being continued and an effort will be made to obtain more data on natural cases of poisoning in swine.

SUMMARY

Ground *Crotolaria spectabilis* Roth seed were fed to five hogs in 2-gram daily doses and to ten hogs in 0.5-gram daily doses with fatal results in most cases. Whole seed of *C. spectabilis* administered in capsules passed undigested through the alimentary tract of experiment animals. The outstanding gross lesions of *C. spectabilis* poisoning in swine were severe anemia, accumulations of fluid in the abdominal and thoracic cavities, ecchymoses of the endocardium, and gastritis, death often being caused by gastric hemorrhage. The most pronounced microscopic lesions were passive congestion, cloudy swelling of all the parenchymatous organs, heart muscle and smooth muscle of the viscera, edema, and foci of necrosis in the lymph-nodes and parenchymatous organs.

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The mouse that hath but one hole is quickly taken.

George Herbert.

There is no wisdom like frankness.—*Disraeli.*

SOME OBSERVATIONS ON FILARIASIS AND ITS TREATMENT*

By I. M. HAYS, *Albany, Ga.*

In the beginning of these experiments, the disease had assumed widespread and serious proportions among dogs. The only treatment that had been used with any degree of success was the organic arsenicals, chief of which were neo-arsphenamine, atoxyl and various cacodylates. In March, 1932, I began the use of both atoxyl and neo-arsphenamine on all my clinical cases. The result seemed to be encouraging; the general condition was materially improved and in some cases there appeared to be some decrease in larval content. After continued use of the arsenicals, it became obvious that the apparent good results were merely the highly tonic effect of the drug; the parasites were in no way affected. With good food, good care and proper medication, it is possible to build up the general resistance of the dog over and above the debilitating action of the parasites.

Palliative measures and temporary relief are of little or no value to field trial and shooting-dog kennels. Consequently, in April, 1932, experiments were started to attempt to find a treatment that would actually destroy the parasites. The experiments were most difficult and costly in the beginning, since the only apparent method was to select some likely drug and inject it into a dog experimentally. The drug injected, of course, destroyed many dogs. A kennel in Port Chester, New York, had lost several dogs from filariasis, and especially bitches at whelping time. The entire kennel was shipped to me to dispose of as I saw fit. Two of the bitches were extremely heavily infested and had almost died in whelping. A microscopic blood examination was made each day over a period of three months and the estimated number of larvae for each drop of blood examined was 127. These cases were given neo-arsphenamine in proper dosage and they showed a nice improvement in general condition but there was no effect on the number of larvae. It seemed desirable to do some direct work on the blood and this very heavily infested blood was particularly ideal. It was found that when the blood was properly treated with potassium oxalate the larvae remained alive and showed normal activity for twelve to 48 hours. We were then able to place a drop or two of blood on a slide, add to

*Presented at the joint meeting of the North Carolina State Veterinary Medical Association and the South Carolina Association of Veterinarians, Spartanburg, S. C., July 10-11, 1934.

it some of the chosen drug and observe with the microscope the action of the drug on the larvae.

The drugs were classified and divided into groups according to their therapeutic activity. Since heart-worm disease is reputedly mosquito-borne and the infestation transmitted much the same as malaria in the human, it seemed logical to try the cinchona group first. No drugs of this group were of any value. The old and effective drugs of the anthelmintic group were of no value. It was interesting to note that the larvae would swim through small globules of both tetrachlorethylene and oil of chenopodium without visible effect.

The first group found to be destructive to the larvae was the volatile oils, and of this group, oleum hedeoma was very lethal, with oil of citronella running a close second. The hedeoma was so powerful it produced death of the parasite almost immediately. A stiff-jointed probe could be dipped into the oil, the excess oil shaken off, then dipped quickly into 10 cc of infested blood, and in ten minutes all larvae in the blood would be dead.

Antimony Gives Encouraging Lead

With direct microscopic examination of the blood, many drugs could be tried in one day. Next antimony, tin, bismuth and arsenic preparations were tried, and of these antimony was found to be the most valuable. The antimony preparation used was tartar emetic. It was much slower in action than the oils, but destruction was just as complete.

The problem then became rather more difficult—the transfer of the experiment back to the living animal. The oils were tried first, prepared in different ways and given intravenously. Oleum hedeoma produced disturbance in locomotion and clotting of the blood in the vein as far up as it was possible to palpate. The oil of citronella produced infarcts in the lungs and resultant pneumonia. When we found the oils could not be safely given intravenously, we started injections of tartar emetic. The first test case to which it was administered was a hound heavily infested and greatly debilitated. Two grains were given intravanously on May 2, 1932. The next day the case was greatly depressed but there was little change in the number of larvae. However, their movements were slower.

On May 4, the dose was repeated. On May 5, the blood was clear of larvae, the dog was in the last stages of debilitation and it appeared to be a fatal case. The blood was very thin and had a peculiar brownish color. The dog was given large doses of sodium thiosulfate and otherwise stimulated. Eventually, with

careful nursing, it made a recovery. In two weeks the dog was sufficiently recovered to go home and has been perfectly normal for the past two years, with no reappearance of the larvae. It seemed from this that tartar emetic might offer possibilities. Many other dogs were injected with it but with discouraging results.

Occasionally a case responded nicely, but it was a very common thing to find the dog dead the morning after having been injected with tartar emetic the night before. Small doses failed to destroy the parasites. If the dose was sufficient, there was danger of either sudden death or severe lingering symptoms, which consisted of chronic enteritis, nephritis, and quite often jaundice. It then appeared probable that tartar emetic was lethal to the parasites but could not be tolerated well by the host. It was thought that the potassium contained in the tartar emetic might possibly be the effective agent. Various preparations of potassium were injected in test cases with no effect on the parasites. Therefore, it seemed that antimony was responsible for the destruction of the parasites.

Tartar Emetic Unsafe for Use

It was found that tartar emetic could neither be sterilized by heat, nor could it be alkalinized; in each instance, a precipitate occurred. Tartar emetic was used on many experimental and even clinical cases for a period of three months. The drug was obtained from many sources in order to determine, if possible, any variation in its potency or toxicity. There appeared to be little, if any, difference. Enough dogs were used in this work on tartar emetic to determine definitely that antimony was highly specific in its action on the parasites, but the particular salt tartar emetic could not be tolerated by the dog and it was entirely unsafe to use as a routine treatment.

All dogs that died during treatment were carefully autopsied and a search made for adult worms. In all heavily infested cases that died after the first or second injection, the adult parasites were found. In some of these cases the adults would be found twisted together like a rope in one of the larger vessels, especially one of the pulmonary veins, or they would be tightly wadded together in a cavity of the heart. In many of these cases the parasites in the center of the rope, or ball, were alive, while those on the outside were partially disintegrated. In less heavily infested cases that died during treatment, and especially those where the larvae had disappeared from the peripheral circulation for some time before death, the adult worms were seldom found.

If they were present, they were found to be in various stages of decomposition. Dogs that recovered from the antimony treatment were destroyed and autopsied at varying periods after recovery, ranging from five days to a month, and in only one instance were the adults demonstrated.

After it was definitely determined that antimony had a very specific action against both adults and larvae, and that tartar emetic was entirely unsafe, it seemed desirable to attempt to prepare an antimony product that could be tolerated by the animal and still be lethal to the parasites. After months of work, a double antimony salt was evolved which is tolerated well by the animal and appears to be even more destructive to the parasites than tartar emetic. It can be made to carry any pH desired. It should be given intravenously. It is tolerated as well as any other antimony preparation when given subcutaneously or intramuscularly, but it has the antimony characteristic of producing heavy indurations and abscess formation occasionally.

Rapid Accumulation of Antimony a Factor

The primary factor in ridding the blood-stream of filaria seems to be a rapid accumulation of antimony in the blood, and it is possible to do this only by the intravenous method. Since the treatment has been perfected, it has been used in my clinic on hundreds of cases successfully, and the mortality rate is very low, depending on how carefully the cases are selected, ranging from 2 to 4 per cent.

To treat heart-worms in dogs successfully, one should be thoroughly familiar with the disease and should know something of the physiology of the dog. It is different from any other disease we are called on to treat in veterinary medicine. In this case we have large parasites, many of them 16 inches long, actually within the blood-vascular system, and with no avenue for expulsion. In the case of milk fever, we have a rather specific treatment which we may inject and reasonably expect the injection to do all the work, but in the case of heart-worms, the treatment is just as specific but it is not sufficient simply to make the injection and expect the treatment to do all the work.

No dog has ever recovered from heart-worm disease without undergoing a reaction, the severity of which depends largely on the number of parasites and especially the adults present. It must be remembered that when these large worms are killed they must be broken down and eliminated. When they break down, this suddenly releases a vast amount of toxic foreign material in the blood-stream. If a dog is allowed to become severely in-

fested and the general debility is great, the breaking down of a vast number of adult worms simply produces more toxic material than can be eliminated successfully, and the death of the animal results.

In a few cases we find dogs that are sensitive to antimony, and poisoning results, but such cases are rare. A large number of non-infested dogs have been given therapeutic injections of the antimony treatment over a period of time and only in rare instances is there any noticeable effect, and then the reaction is vastly different from that seen in infested cases. It should be a policy to advise all clients that the earlier in the disease the case is treated, the better the chance of recovery. The chance of recovery is in direct proportion to the number of adult worms present.

How Are the Parasites Eliminated?

The question is often asked as to how the parasites are eliminated, and of course that cannot be answered as yet. It is known that 85 per cent of the adult is fluid, and the framework is of a fibrous nature. As soon as the adult dies, the skin ruptures in many minute places and the fluid "boils" out. Under a magnifying glass the surface appears to be covered with countless boiling volcanoes. It is possible to watch a larva in oxalated blood under the microscope until it dies. As its movements become slower, small transparent globules are seen to attach themselves to it, and when all movement ceases, the parasite is entirely covered with those globules. Upon staining the slide, these globules prove to be leucocytes of various types. It is therefore assumed that the fluid part of the parasite is eliminated chiefly through the kidneys and the framework is eventually carried away by leucocytic action.

The two bitches whose blood was used in the first experiments were treated successfully, sent home, and were normal in all respects. They both whelped normally in the past two years and both did normal work in the field. One of them was sent to my clinic two months ago for observation. The mucous membranes were cyanotic and the heart action was very rapid and feeble. She died suddenly. An autopsy was made and the heart was found to be enlarged to twice its normal size, the lungs showed severe passive congestion, and a large thrombus was found in the largest of the pulmonary veins. It was an inch in thickness and was six inches in length. It had many branches and extended to many parts of the lung. The thrombus was of a red-

dish-brown color, was firm and dehydrated, and had been deposited in distinct layers. In addition to this a severe phlebitis was present, small transparent tumors were attached to the heart-valves, and a small ulcer had penetrated through the wall to the epicardium of the right antrim. No filaria were present.

Another such case was found some months after treatment. It is reasonable to suppose that the phlebitis present was the result of the original heart-worm infestation, and the thrombus the result of a remnant of framework of the filaria that had not been completely carried away. It seems evident that any case that is extremely heavily infested over a period of time may be left with a cardiac deficiency even though the parasites may be eliminated successfully. Many of the field-trial and shooting-dog kennels of the South have been treated as long as one and a half years. They have been repeatedly checked since, and in only a few instances have larvae reappeared in the blood, or symptoms of the disease recurred.

Each Case Presents Problems

It is not possible to write a set of directions for all emergencies in the treatment. Each case presents its own individual problems. The most valuable information can be gained only through experience. No heavily infested and debilitated case should be given a favorable prognosis, and it is a safe procedure to treat cases only at the owners' risk, since we find that the number of larvae is not always a good criterion of the number of adults that may be present.

The first thing in the treatment of any case is to give a thorough examination. Investigate the possibilities of intestinal parasites, and make a close examination of the quality of the blood and heart action. Take into consideration the age of the dog and the possibility of any secondary disease that may be present in a latent state. If some secondary disease is present when treatment is begun, it will become acute in most instances and cause the death of the dog. It is most important also that the dog have no dietary deficiencies and a good precaution is to make sure raw meat is fed in liberal quantities for a week or two previous to the beginning of the treatment. Meat should be fed during and immediately following the treatment.

The technic of injection is simple. Compress the front leg at the elbow and raise the large cephalic vein that passes up the front of the forearm, fill the syringe and leave the needle attached. Insert the needle into the vein until a free flow of blood passes into the syringe, then inject rather rapidly. The vein in the

front leg is preferred over the external saphenous for the reason that the treatment will mix with less blood before it comes in contact with the adult parasites, which are usually situated in the right heart or pulmonary vessels. In our later work we find some evidence to support the idea that in the majority of cases the adult worms are destroyed before the larvae completely disappear from the blood-stream.

The size of the dose and the frequency of its repetition is most important. It is our common custom to keep a case under observation and treatment, if necessary, for several days before the direct heart-worm treatment is administered. It is quite necessary to make sure that the dog is in the best possible condition and ready to receive the treatment. In heavily infested and debilitated cases, it is often necessary to give a build-up treatment for several weeks before the antimony injections are attempted. We usually give 3 cc as an initial dose to a dog the size of a mature pointer. The injections are repeated at intervals, according to the reaction of the animal and the action of the larvae. The average case will take an injection every second day. A few cases must have it every day, and on the other hand, the injections must be spaced farther apart in some dogs.

Observe the Larvae

An important part of the treatment is close observation of the larvae. As the antimony accumulates in the blood, the movements of the larvae become markedly slower. There is a critical point in the series. When the larvae become very slow in their movements, and if a rather large dose can be safely given at this time, the larvae rapidly disappear. If, for some reason, the injection is omitted at this time, the antimony is rapidly eliminated and the parasites regain their normal activity. It is necessary in this case to start the series of treatments all over again. We believe in increasing the dose as much as possible and pushing the treatment until a reaction occurs. We give 3 cc the first dose, 4 cc the second, and often give a large dog as much as 5 cc. The maximum therapeutic dose and the minimum lethal dose have not been determined and we do not believe it possible to establish them.

We do not always wait for a reaction to subside before giving the next injection. It all depends on the type of reaction. The symptoms most commonly seen in a reaction are, first of all, a loss of appetite, vomiting, transient diarrhea, acute rheumatism, myositis or arthritis, and often a type of anaphylaxis in which

the head and nose are swollen and the eyes protrude. In any type of reaction, the pulse rate becomes very fast and often very weak. All of these reactions must be handled symptomatically. Sodium thiosulfate in one-half-ounce doses is of value. The intravenous injection of glucose (10 cc) and rather large quantities of physiologic salt solution have proven of value. Plenty of exercise all during the treatment and especially during any type of reaction is quite necessary.

Some Cases Require Second Treatment

We have observed a certain type of blood quality in which the treatment seems to have as much affinity for the blood (hemoglobin or white cells) as for the parasites. This type of case often requires a second series of treatments. On the other hand, we have observed another type of blood quality in which the treatment has little or no affinity for the blood and the parasites are destroyed most rapidly. We are unable, as yet, to say what constitutes that difference. Occasionally a case occurs where the larvae rapidly disappear and then recur at periodic intervals and readily disappear again under treatment. We were fortunate enough to be able to post two such cases and found in each case the adult parasites encysted in tissue outside the blood-stream.

In one case the parasites were in an enlarged cyst in the thoracic aorta, with a direct opening to the lumen of the artery. In the other case the adults were in cysts in the lung tissue, with direct openings into the blood-stream. In both cases they could continue to give birth to the living larvae with complete immunity from the treatment. Two types of cases then are sometimes encountered where the treatment is apparently not effective against the adults, one being the case where they are outside the blood-stream and the other being the ocasional, heavily infested case, where they are tightly rolled together and partially or completely occlude some vessel. In this case the treatment does not freely circulate through them.

Much remains to be done in an experimental way. The surface has hardly been scratched. The microscopic examination of blood for the purpose of making a diagnosis is very crude and the chance of error is very great. It seems possible to develop some sort of allergic reaction, or possibly a systemic or temperature reaction that would be more accurate. Further studies may also reveal the exact composition of the blood which is responsible for the vast difference in the amount of treatment required in average cases. It seems possible also to find a method of favorably altering the blood for the most successful treatment.

Heart-worm disease is a most serious disease in dogs. It is rapidly spreading and will continue to do so until veterinarians and clients alike realize the seriousness of it and combine their efforts to halt the advance. Every infested dog is a carrier and potential spreader, and remains so as long as he goes untreated.

Shortage of Veterinarians in England Only Temporary

That the shortage of qualified men in the veterinary profession in England is but temporary, is the opinion of Dr. Fred Bullock, secretary of the Royal College of Veterinary Surgeons, in a communication to the *Lancet*, of November 10, 1934. According to Dr. Bullock, the number of admissions to the profession is rising steadily. The number of students in training at the five veterinary colleges in England is the highest ever recorded, and is sufficient to provide a graduation figure for the next five years double that of losses by death or otherwise.

Bang's Disease Tests Reach High Figures

Early in December, the U. S. Bureau of Animal Industry reported that the testing of cattle for Bang's disease was under way in practically all states. During the months of July, August, September and October, 232,185 cattle were tested, of which 33,368, (about 14 per cent) reacted to the test and were designated for removal and slaughter. It will be noted that the actual results during the period were very closely in line with the estimates that had been made by veterinarians in the Bureau of Animal Industry. Before the work was started, it was believed that reactors would run around 15 per cent.

In each of four states—Virginia, Ohio, Minnesota and Wisconsin—more than 20,000 cattle were tested. Minnesota was reported to have 360,000 cattle on the waiting list. The total number of cattle on all waiting lists throughout the country on October 31 was 891,968. Dr. A. E. Wight, who is in charge of the campaign for the federal government, expressed the opinion that, in view of the large number of cattle on the waiting lists and the preparations that were being made for pushing the work in a number of states, the number of cattle that would be tested during December would probably establish a record. It is planned to publish monthly statistical reports on the progress of the campaign to eradicate Bang's disease.

MYCOTIC AFFECTIONS OF THE BOVINE REPRODUCTIVE SYSTEM*

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Aside from the outstanding importance of Bang's abortion disease, disturbances in the reproductive system of cattle have been attributed to trichomoniasis, streptococcosis, vibrionic abortion, mycosis, deficiency diseases, and toxemias. Brucella-negative cases in which the etiologic factor is unknown are loosely grouped under the term "sporadic abortion." Since deliveries before term or other symptoms of disturbed sexual health occur not infrequently in herds known to be free from Bang's abortion disease, an etiologic analysis of such cases is highly desirable from both the scientific and practical standpoint, in furtherance of any major program in the control of genital diseases. During the past three years, several cases of mycotic affections of the reproductive system were brought to the attention of this laboratory as the official Connecticut laboratory for the establishment and maintenance of cattle herds free from Bang's abortion disease. A study of these cases is presented here.

LITERATURE

What appears to be the first case of mycotic infection of the fetal membranes in cattle was reported by Smith[1] as occurring in a pregnant uterus secured from an abattoir; the intactness of the uterine seal precluded the possibility of secondary infection. The cotyledons showed crater-like central necrosis; bacteriological and pathogenicity tests for *Brucella abortus* were negative, but the presence of a mold-like organism was demonstrated by cultural and histopathologic means in the chorion, amniotic fluid, fetal lungs, and digestive tract. In artificial culture the organism exhibited abundant whitish aerial mycelium, rhizoids and sporangiophores, sporangia of about 80x64μ dimensions, a pear-shaped columella and spherical spores 4$\mu\pm$ in diameter, and double-contoured chlamydospores. It was thought to resemble *Mucor rhizopodiformis* Lichtheim,[2] which is now known under the name of *Rhizopus cohnii*, according to Henrici.[3] Carpenter (quoted by Gilman and Birch[4]) reported the isolation of a mold in pure culture from the digestive tract of a 7-month-old aborted

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fetus; the botanical position of the organism was not determined. Gilman and Birch recovered a Mucor which was practically identical with that described by Smith[1] from two aborted fetuses about six months old, and were able to produce placental infection experimentally in two out of five cows inoculated with it. The three cases cited occurred at intervals of approximately one year in a herd which was free from Bang's abortion disease, according to the serological record.

Probably the most extensive study along this line has been carried out in Denmark. Bendixen[5] observed a case of generalized mycosis due to *Aspergillus fumigatus* in a cow, and was later[6] able to show that placental infection could be produced experimentally with this organism. Bendixen and Plum[7] reviewed the literature on mycotic affections in cattle. They found, in the course of the examination of placental material from aborted fetuses, 17 Brucella-negative cases, of which eight were infected with *A. fumigatus* Fresenius, two with *Absidia ramosa* Vuillemin (syn. *Mucor ramosus* Lindt), and seven with both of these organisms. The mycotic abortions occurred during the second half of gestation. Experimentally it was shown that the normally saprophytic but potentially pathogenic molds grow best at 37° C., that they can be isolated readily on liquid media, and that placental specimens artificially infected with molds do not exhibit evidence of growth in less than 60 hours at room temperature. In histologic sections the hyphae of the phycomycetes comprising the genus Mucor appeared thick, often swollen, double-contoured and indefinitely septated; the hyphae of the mycomycetes comprising the genus Aspergillus appeared comparatively thin, usually single-contoured, evenly thick and markedly septated. Of five cows inoculated intravenously with spores of the molds, five showed evidence of mycotic infections and aborted; it was impossible to produce infection *via* the digestive tract, but it appeared that under natural conditions infection may take place through the respiratory tract from which the organisms are carried by the blood-stream into localities of lowered resistance, such as the placental tissue of the pregnant uterus. Plum,[8] in continuation of these studies found among 4,133 placental specimens submitted for examination 1,293 positive for *Br. abortus*, 2,027 negative for this organism, and 695 decomposed and unfit for examination. Of the *Br. abortus*-negative specimens, 121 showed microscopic, but not cultural, evidence of mycotic infection, and 118 yielded growth of hyphomycetes. The collected cultures were classified in coöperation with the Centralbureau voor Schimmelkultures in Baarn (Holland) as follows: *A. fumi-*

gatus occurred 82 times (69 p. c.); *A. versicolor*, 3 times (1 p. c.); *A. flavus*, once; *Ab. ramosa*, 21 times (13 p. c.); *Ab. Lichtheimi*, 4 times (1 p. c.); *Mucor pusillus*, 6 times (4 p. c.); and *Rhizopus bovinus*, n. sp., Van Beyma thoe Kingma, once.[9]

Observations

Case 1: The specimen consisted of the fetus and placenta of a 6-year-old grade Holstein which aborted in the seventh month of gestation. The herd history indicated that all animals had reacted negatively to serological tests for *Br. abortus* during the previous three years and that no abortions had occurred within this time. The cow in question was reported as having produced four normal calves prior to the trouble; she was bred 12-22-30 and again 5-6-31; the last service resulted in pregnancy which was terminated by abortion, 12-2-31; cultural examination of fetus and placenta was made 24 hours after the incident, and yielded a diplostreptococcus; a guinea pig injected intraperitoneally with saline suspension of placental material died 17 days later, showing yellowish nodules in the liver; a diplococcus was isolated from the affected organs; mycological examination was not made. Histologic examination of fetal and maternal cotyledons showed numerous mycelial structures with the general character of Mucor hyphae (fig. 1). Following this abortion the cow was bred 12-24-31, 3-6-32, 3-29-32, and 4-19-32, and gave birth to a normal calf 1-21-33, but retained the afterbirth; she was bred again 3-26-33, 5-1-33, 5-29-33, and 7-22-33, when she was sold for beef on account of sterility. It should be mentioned that the finding of the mycelial structures in placental sections was entirely incidental to a search for evidence of diplococcus invasion. This was the first case that directed the author's attention toward mycotic affections as possible explanations of abortions in Bang's abortion disease-free herds, and the records are admittedly incomplete; the breeding data before the occurrence of the abortion with an unexplained interruption of the sexual cycle for 4½ months (unnoticed abortion ?), the multiple services required for the subsequent impregnation plus the retention of the afterbirth terminating in sterility, are suggestive of the etiologic importance of the Mucor infection observed.

Case 2: The specimen consisted of the fetus and placenta of a 5-year-old Holstein cow which was killed during the fourth month of gestation on account of streptococcic mastitis. The herd in which cases 2, 3 and 4 were observed has been free from Bang's abortion disease since 1925, but has experienced a moderate num-

ber of abortions and sterility troubles throughout the period 1925-1934. The animal was bred 6-24-31, 12-2-31, and calved normally 9-8-32; she was bred 10-1-32, 10-22-32, 11-15-32, 1-9-33, and calved normally 10-18-33; she was bred again 11-8-33 and 11-28-33, and killed 3-12-34. On examination of the intact uterus two hours after slaughter, the chorion-allantois and fetus appeared to be normal; the visceral aspect of the amnion exhibited about 100 irregular, whitish, raised areas 0.5 to 1 mm in height and from 2 to 10 mm in diameter (fig. 2). Histopathologically, the amniotic changes consisted of large spherical to polygonal cells with comparatively small hyperchromatic nuclei; small foci of karyorrhectic changes were observed, but no mycelial structures. Cultural examination of the uterus, chorion and fetus gave nega-

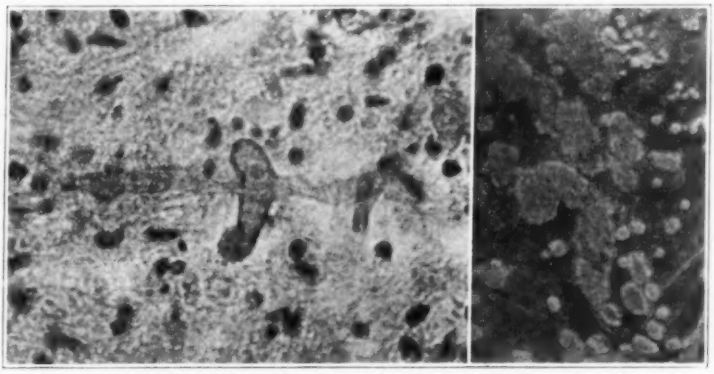

FIG. 1 (left). Necrotic cotyledon (case 1) showing Mucor hyphae (x 800).

FIG. 2 (right). Visceral aspect of amnion (case 2) showing irregular "epithelial thickenings" (actual size).

tive results, but the amniotic changes yielded an organism resembling *A. fumigatus* in pure culture. The amniotic areas resembled the "normal" epithelial thickenings cited by Williams,[10] in location but not in configuration. Another bovine fetus examined at the same age, from a healthy cow, did not show corresponding changes in the amnion. The outcome of the pregnancy which was terminated by slaughter of the dam must, of course, be left to speculation, but the four services required for the production of the second calf would leave room for doubt as to the complete sexual health of the animal.

Case 3: The specimen consisted of the fetus, placenta and uterus of a 4-year-old registered Holstein which was killed 1-2-34, on account of habitual abortion. The animal had been bred 9-27-31, 11-25-31, 7-26-32, 11-29-32 and 12-20-32, and aborted

7-1-33; she was bred again 7-23-33 and aborted 12-31-33. On examination of the uterus, which was performed two hours after slaughter but 48 hours after the actual abortion, numerous hemorrhagic, necrotic cotyledons presented themselves covered by yellowish fibrinous exudate; in sections large focal necrobiotic changes dominated the picture, but mycelial elements were not demonstrated. Cultural examination of the fetus and placenta failed to reveal any significant organisms, but mycologic examination of the necrotic cotyledons yielded organisms resembling *A. fumigatus, A. niger* and *Rh. cohnii,* the last mentioned predominating. Healthy-appearing cotyledons were either sterile or yielded *A. niger.* While the breeding history is indicative of the importance of the mycotic infection, the comparatively long time elapsing between the breaking of the uterine seal and the cultural examination leaves open the possibility of extraneous contamination.

Case 4: The specimens consisted of fetus and placenta aborted in 1931, and fetus and placenta aborted in 1933, from a 10-year-old registered Holstein cow which was finally killed on account of chronic mastitis. The animal was bred 1-27-26 and calved normally 11-9-26; she was bred 1-18-27, 2-10-27, 3-18-27 and 4-7-27, and delivered a mummified fetus; she was bred 1-25-28, 3-10-28 and 3-30-28, and calved normally 12-31-28; she was bred 3-7-30 and calved normally 12-17-30; she was again bred 4-22-31, and aborted 12-4-31; Mucor mycelium was demonstrated in placental sections. She was again bred 2-11-32, 3-2-32, 4-19-32 and 5-30-32, and calved normally 3-4-33, and again 3-21-33, 5-8-33, and 5-30-33, and aborted 12-11-33. The fetus and uterus were examined three hours after abortion. Many of the cotyledons showed extensive dry, leathery, craterlike, central necrosis surrounded by a wall (fig. 3) and were marked by great irregularity in size. Histologic sections of necrotic cotyledons showed evidence of heavy infection with mucor-like mycelial elements; cultural examination of the fetus and placenta was negative for any significant bacterial organisms, but a mold resembling *M. pusillus* was isolated in pure culture from the placental tissue. This case was particularly interesting because a fairly normal reproductive cycle was interrupted by an abortion in which evidence of mycotic infection was obtained; although the subsequent pregnancy took a normal course, it required multiple services; the succeeding pregnancy terminated again in abortion and evidence of mycotic infection was obtained by cultural and histologic tests.

A summary of the data is presented in table I.

TABLE I—*Summary of data on mycotic infections of bovine uterus.*

CASE	ANIMAL	REASON FOR DISPOSAL	CONDENSED BREEDING HISTORY	EVIDENCE OF MYCOCIS		ORGANISMS ISOLATED	
				CULTURAL	HISTOLOGIC†	SPECIES	PATHOGENIC FOR RABBITS
1	Grade Holstein 6 years	Sterility	3 normal calvings 2 services; aborted 7th month 4 services; calved, retained placenta 4 services; sterile	.....	Placenta +		
2*	Holstein 5 years	Mastitis	2 services; normal calving 2 services; normal calving 2 services; killed 4th month pregnancy	Amnion +	−	*A. fumigatus*	+
3*	Holstein 4 years	Abortion	5 services; aborted 7th month 2 services; aborted 5th month	Placenta +	−	*A. fumigatus* *A. niger* *Rh. cohnii*	+ − +
4*	Holstein 10 years	Mastitis	1 service; normal calving 4 services; mummified fetus 3 services; normal calving 1 service; normal calving 1 service; aborted 8th month 4 services; normal calving 3 services; aborted 6th month	..... Placenta +	Placenta + Placenta +	*M. pusillus*	+

*From same herd.
†Fungous elements in sections had the character of Mucor hyphae.

Description of Organisms

The two species of Aspergillus isolated from cases 2 and 3 grew readily on Sabouraud's agar and modified Czapek's solution agar at 37° C. and at room temperature.

The aspergillus-like organism isolated, in common, from cases 2 and 3 produced green to dark green columnar heads on smooth stalks; the vesicle was globose, and the single row of sterigmata carried tightly massed chains of spherical conidia about 3μ in diameter. The organism was pathogenic for rabbits on intravenous injection, causing death in about four days; on postmortem examination the kidneys and liver showed nodular lesions, and the organism could be recovered from the internal organs. In sections the kidney lesions presented primarily the picture of

Fig. 3. Part of uterus (case 4) showing enlarged cotyledon with dry central crater-like necrosis (one-third actual size).

glomerular nephritis (fig. 4) and under high-power magnification numerous single-contoured mycelial hyphae (fig. 5) were demonstrated. According to the classification of Thom and Church,[11] the organism falls into the *A. fumigatus* group; since perithecial structures were not observed, a definite placing of the organism in the group was not undertaken.

The other aspergillus-like organism isolated from case 3 produced large, dark-brown, globose heads on smooth stalks; the double row of sterigmata carried spherical, brownish, slightly tuberculated conidia about 5μ in diameter; sclerotia were not

observed. The organism was not pathogenic for rabbits. According to the classification of Thom and Church, it falls into the *A. niger* group.

The two species of mucor-like fungi isolated from cases 3 and 4 grew readily on Sabouraud's agar and moistened white bread at 37° C., but poorly at room temperature.

The organism obtained from case 3 produced at first a whitish, later a grayish, tall, fluffy growth which spread over the substratum. A bread culture attained an apparent height of from 2 to 6 cm, if allowed to expand; it did not show evidence of geotropism. The branches of the rhizoids in the substratum were straight and thorn-like; stolons were often indistinguishable from other mycelial structures such as sterile hyphae, but could be demonstrated in inverted Erlenmeyer flasks where they formed attachments to the glass wall; sporangiophores rose from the nodes of the stolons, either single or in fascicles, and showed secondary, occasionally circinate, branches. The sporangiophores were erect or slightly curved, about 130μx10μ in dimensions, and formed a funnel-shaped thickening below the terminal sporangium, a so-called apophysis. The sporangia were spherical to piriform, from 50 to 90μ in diameter, and the sporangial wall was finely incrustated and diffluent in the presence of water. The columella was subjacent and (together with the apophysis) oval to piriform, occasionally deltoid, in shape, and about 30x40μ in dimensions; the membrane of the columella was smooth and colorless showing occasionally a line of detachment but no distinct colarette. The spores were spherical to oval, 4 to 5μ in diameter, smooth and colorless. Occasionally, sporangiola without a columella and containing from seven to nine spores, were observed on secondary sporangiophores, especially in old cultures. Bizarre, double-contoured giant-cells resembling the swollen club-shaped elements seen in animal lesions (fig. 1) were observed in protein-rich substrata; chlamydospores or zygospores were not recognized. The fungus was pathogenic for rabbits on intravenous injection, and did not lose its virulence in serial transmissions; while the gross lesions resembled that produced by *A. fumigatus*, the alterations in the kidneys presented the microscopic picture primarily of tubular nephritis (fig. 6), and under high power the mycelial elements were coarse, double-contoured, and often swollen and branched (fig. 7). According to Fischer's[12] and Lendner's[13] keys, the organism resembled *Rh. cohnii* Berleese et de Toni.

The third fungus isolated from case 4 produced at first a dark gray, later a brownish growth which became very dusty in

appearance on aging; cultures did not attain an apparent height of more than 3 mm. The rhizoids showed multiple branching in the substratum; sporangiophores arose directly (fig. 9) from the

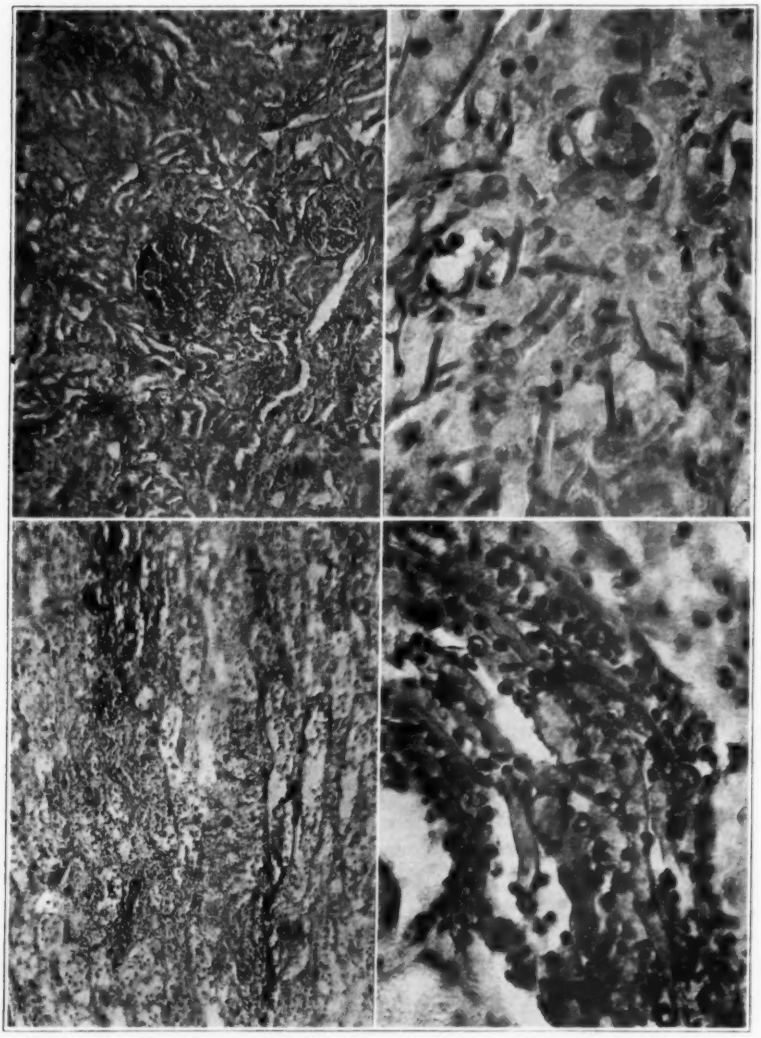

FIG. 4 (upper left). Kidney of rabbit artificially infected with *A. fumigatus* showing primarily glomerular nephritis (x 100).

FIG. 5 (upper right). Liver of rabbit artificially infected with *A. fumigatus* showing character of mycelium (x 800).

FIG. 6 (lower left). Kidney of rabbit artificially infected with *M. pusillus* showing primarily tubular nephritis (x 100).

FIG. 7 (lower right). Kidney of rabbit artificially infected with *M. pusillus* showing character of mycelium (x 800).

medium, and stolons were not observed. The sporangiophores were erect, about 1 to 1.5 mm in height and from 6 to 13μ in width, at first unbranched, later showing grape-like racemic branching; all sporangiophores were terminated by spherical, multispored sporangia, 60 to 90μ in diameter, which became brownish on aging; the sporangial membrane was incrustated by fine calcium oxalate crystals, and diffluent in the presence of water. The columella was spherical to ovoid, free (not subjacent), and from 40 to 50μ in diameter, its membrane colorless and smooth; a small colarette was sometimes observed. The spores

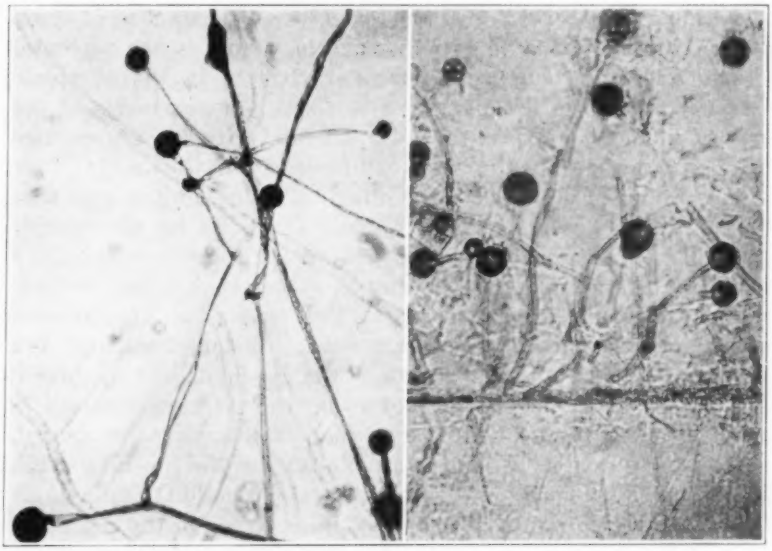

FIG. 8 (left). Slide culture of *Rhizopus cohnii;* note stolon, spherical sporangia and subjacent columella with apophysis (x 100).
FIG. 9 (right). Section of culture of *Mucor pusillus;* note direct emerging of sporangiophores from substratum, spherical sporangia and free columella (x 100).

were round, about 3.5μ in diameter, smooth and colorless. Occasionally double-contoured, circular oidiospores were seen on the aerial mycelium, and swollen giant-cells in the substratum. Chlamydospores and zygospores were not recognized. In its pathogenic action and morphology of hyphae *in vivo*, the organism corresponded to *Rh. cohnii* of case 3. According to Fischer's[12] and Lendner's keys,[13] it more closely resembled *M. pusillus* Lindt.

DISCUSSION

The recognition and analysis of the etiologic factors responsible for cases of so-called sporadic abortions should be an integral part

of any organized campaign against Brucella infections and, in a wider sense, against genital diseases in general in the bovine species. This is desirable not only from the standpoint of eliminating, or at least restricting, the use of the indefinite and unscientific term "sporadic abortions," but possibly more so from the viewpoint of the owner and practitioner, who are interested in reaching the promised land of undisturbed reproductive capacity in a herd. Among the etiologic factors of infectious abortions in animals free from Bang's disease, various fungi of the types Aspergillus and Mucor have been considered. From transmission experiments carried out by Gilman and Birch[4] and by Bendixen and Plum,[7] it appears fairly well established that molds of these types have the power of invading and localizing in the placental tissue, and of producing severe disturbances in the chorionic attachments of the pregnant uterus. That common molds of the type of *Rhizopus nigricans* are also endowed with toxic properties has been known since the work of Blakeslee and Gortner.[14]

The avenue of entrance of the infection is unknown, except that the possibility of the digestive tract seems to be eliminated; available evidence would point toward a primary respiratory infection metastasizing in the uterus, rather than a local ascending affection of the genital tract. The present study was concerned mainly with the correlation of pathologic, cultural and histologic findings in the placenta on the one hand, and the breeding history on the other. It seems that the evidence obtained in case 4 of mycotic infection, in 1931 and 1933, in the same animal, accompanied by a definite unfavorable turn in the breeding cycle, would indicate a persistence of the mycotic infection in an animal, it thereby becoming an *important disease factor* in the reproductive cycle of the *individual.* An early diagnosis of such cases which, it is felt, is best established by histologic methods, may also open up the possibility of specific chemotherapy by iodides and perhaps other chemical agents.

The multiplicity of fungous organisms, coupled with the apparent lack of epizoötic progress on a premise, would indicate that mycotic infections of the genital tract are of *secondary importance,* from the standpoint of *herd sanitation.*

SUMMARY

During the examination of placental tissues from three cases of spontaneous abortion and one case of abnormal epithelial thickenings in the amnion, in herds free from Bang's abortion disease, mycotic infections were demonstrated by cultural or histologic means.

In one animal evidence of mycotic infection was obtained on the occasion of two abortions which occurred two years apart.

A correlation of the breeding data and laboratory findings signifies the etiologic importance of such infections.

The organisms implicated in these cases resembled *Aspergillus fumigatus, Aspergillus niger, Rhizopus cohnii* and *Mucor pusillus.*

Mycotic affections of the pregnant uterus seem to be of intrinsic importance with regard to the individual sexual health of the animal, but of only slight importance from the epizoötic standpoint in the herd.

Cases of abortions of unknown etiology should be made the subject of laboratory analysis as a part of the general program of combating Bang's disease and of other diseases of the genital tract which are of economic importance.

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Danger in Poisons Used as Sprays

Nearly one-third of the time, money and effort expended by the federal Food and Drug Administration is being devoted to protecting the public from the danger of poisons used in sprays to combat insect pests and diseases that attack fruits and vegetables, according to the report for the year ending June 30, 1934.

RABIES CONTROL FROM THE STANDPOINT OF THE HEALTH OFFICER*

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For a medical man to appear before a veterinary group to discuss a disease, with the manifestations of which you are far more familiar than am I, might at first sight appear presumptuous, were it not for the fact that this disease, rabies, may at times have a most important bearing upon the well-being of the human. In fact, it is this possible transfer to the human that gives to rabies its importance even from the veterinary point of view.

Were rabies a disease merely of the dog, or even a disease capable of transmission to all mammals except man, it would attract but little attention. It would not merit so much as a small fraction of the attention that is given to such diseases as hog cholera, contagious abortion or foot-and-mouth disease, for it has no economic importance aside from the occasional loss of a cow or other domestic animal bitten by a rabid dog.

However much the dog-owner may cherish and value his pet, rabies at its worst is a rare enough infection to merit but little attention. Yet the possibility of transference of this disease to man, and the awful certainty of death if the infection develops, have given to rabies an importance out of all proportion to the frequency of the condition. When we then realize that means are at our disposal for the virtual elimination of the disease if we can only show sufficient ingenuity in applying our scientific knowledge, it is easy to understand why rabies offers such a challenge to the health officer, and often occupies such a high place in his scale of values.

It is true, of course, that while rabies is an infrequent disease in dogs it is an extremely rare disease in humans. In the Commonwealth of Massachusetts, numbering over four and a quarter million persons, there have been only eight human deaths from rabies during the past five years. Automobiles kill more in a single week. Why, then, the attention that we give to this rare disease? The reasons are many, all of them revolving around the certainty of death if the disease is allowed to develop. Let

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us, therefore, consider briefly what are the facts with regard to this disease.

How many dogs there may be in a state such as Massachusetts I do not know. If I were to judge from my own neighborhood, I should say almost as many as there are families. Owing to the failure of many communities to enforce the dog-licensing law, all figures from such sources are notoriously below the real facts. I do know, however, that in an average year over 6,000 persons are bitten by dogs and that for the current year the figure will reach almost 8,000. Even these figures are an underestimate of the situation, for although all dog bites, whether or not requiring antirabic treatment, are reportable, it is well known that many of those reaching medical attention are not reported, and even more incomplete is the reporting of those given home care. Any factor in our social organization that injured over 6,000 persons a year might logically merit the attention of the health officer, even without the superimposed threat of rabies, but we will ignore that aspect of the situation on the theory that the pleasure of the dogs' company is worth 6,000 bites, even though some of them be on the face with resultant disfiguring.

Few Dog Bites Are Infective

It should not, of course, be presumed that all of these bites require antirabic treatment. As a matter of fact, very few of them merit such drastic measures. The majority are accidental or are the result of provocation by the individual bitten, frequently a tin-can-minded small boy. An occasional bite is due to an inherent viciousness on the part of the dog. In most instances, therefore, rabies can be excluded through nothing more than a reasonable period of observation. On the other hand, there is a small group bitten by or intimately exposed to dogs proved to be rabid. In an average year, there are about 250 to 300 such dogs in this state. Another group is composed of those bitten by dogs as to the condition of which there may be uncertainty, and finally a group of not inconsiderable size bitten by stray dogs. In 1933, the State Department of Public Health was obliged to recommend treatment for almost 1,000 persons.

Here we have then a problem of no small magnitude. To be sure, some of these persons took the treatment unnecessarily; but who are you or I to determine with certainty who shall need treatment? If we make a mistake in assuring an individual that treatment is not necessary, the result may be fatal. We need only to remember that in the pre-Pasteurian days, bites by rabid dogs

usually resulted fatally in about 16 per cent of the cases, and there is no proof that there has been any attenuation of the virulence of the virus since those days. We, therefore, err on the side of safety and recommend treatment on a somewhat lavish scale, even though we know that in so doing we are subjecting many persons to an unnecessary treatment, which is at best unpleasant. Such is perhaps the most charitable adjective that can be applied to the negative aspects of a treatment which is at the same time life-saving.

There can be no argument as to the value and effectiveness of the so-called Pasteur treatment. To that extent, we have a measure for controlling the disease in humans. On the other hand, the unpleasant and at times even dangerous aspects of the treatment justify some restraint upon our lavish recommendations. The injections are often painful, and paralytic and encephalitic complications are encountered. I can think of nothing more sobering than a death from rabies vaccine given to a person bitten by a stray dog. Who knows whether or not that dog was actually rabid? We know only that on the law of chance there is a greater risk from a bite by a stray dog than in the antirabic treatment, especially if rabies is existent in that area. Consequently we continue to recommend such treatment, each time murmuring a silent prayer that this will not be the one case in several thousand to develop a vaccine reaction.

Such then is the problem of rabies today. A rare disease to be sure, but so awful in its manifestations and so certain in its outcome as to compel us, if exposed, to resort to measures which cannot be described as devoid of danger. To those who elect to "take a chance," there is the awful uncertainty entailed by several months of waiting. It is little wonder then that the public and the health officer accord to this disease an importance out of proportion to its prevalence.

Theoretically, there is no reason why rabies cannot be completely controlled. We have in our possession all the knowledge necessary to achieve this end, so that it now remains to put this scientific knowledge to work. As with all other diseases, our control measures fall into three groups: first, those designed to prevent the spread of the virus from the infected to the non-infected; second, those seeking so to raise the level of resistance of the non-infected that the virus, if it spreads, may not produce disease in the new host; third, those measures designed to minimize the ill effects of those infections that have occurred in spite of all precautions. The latter of this group we have already con-

sidered in its application to rabies, so that we may dismiss it without further comment.

What measures may reasonably be taken to minimize the possibility of spread? Surely nothing is simpler in theory but, alas, in practice, how difficult. Restraint orders have been tried for years, and, while they may temporarily contribute to rabies control, they have contributed nothing of a permanent nature in this country. It is true, of course, that in England they were effective, when vigorously applied. Yet in the United States, with such a multiplication of governmental units, coördinated adoption and enforcement of restraint orders sufficient to eliminate rabies seems like a Utopian dream. In the state of Massachusetts alone, there are 355 governmental units involved, and in such matters coöperation seems unknown.

Some Control Measures Accomplish Nothing

Of what use as a measure of permanent control is a restraint order in one community when all neighboring communities permit and even encourage dogs to run at will? When a real-estate agent can rent a summer cottage on the argument of bringing a dog from a community where a restraint order is in force, it can be no cause for surprise that such restraint fails of its purpose. In this particular instance, the dog developed rabies, biting several people and also infecting other dogs. As applied, this particular restraint order accomplished nothing more than to irritate the dog-owner who, because of a sense of civic responsibility, might comply with the regulation. From the point of view of rabies control, it accomplished nothing, for it was not enforced; nor did any of the six adjoining communities attempt to apply similar restraint.

Unless restraint orders can be applied on an area basis, and unless adequately enforced, it is unlikely that much can be expected from them in the effective control of rabies. They may perhaps minimize the spread of the disease. If so, they are worth the effort. If not, they are a delusion serving but to irritate the public since they are too often enacted in response to a sudden and all too fleeting public demand for drastic action to rectify a condition which today seems of paramount importance and tomorrow is as lacking of interest as an advertisement of yesterday's bargain sale.

Even under the best of conditions, when applied over an area and adequately enforced, restraint orders are of but transient value for, when they are no more, the community is just as sus-

ceptible to rabies as before such measures were applied, and the reintroduction of the infection necessitates a repetition of the restraint. Unless we can be certain that there is no possibility of reintroduction of the infection, either from external sources, or from a reservoir in the wild life of the area, we can never hope to control rabies through sole reliance on restraint measures.

In this respect, the application of restraint measures finds a close parallel in the field of human disease. For years, health officers have endeavored to control diphtheria, scarlet fever, measles and whooping cough through isolation and quarantine. I shudder to think of the vast sums that have been expended for this praiseworthy purpose. But, alas, we have still just as much scarlet fever, measles and whooping cough as ever, and diphtheria was not diminished until other measures were applied.

No thoughtful health officer will think of isolation and quarantine as measures on which to place sole reliance in his efforts to control communicable disease; he will think of them merely as weapons to be used in conjunction with other methods, hoping that through their use he may prevent a few cases and delay a few others until the patient is of such age as better to withstand the infection, or perhaps help to lessen the general severity of the disease. Quite similar is the situation of restraint orders for rabies. They are but valuable weapons in our armamentarium, useful if properly wielded, but under present conditions not of such value as to warrant our placing sole reliance upon them.

The second general method of disease control I have defined as measures which have as their purpose the raising of the resistance of the non-infected to such a level that disease will not result should the virus find its way to a new host. We are all familiar with the fact that every infectious disease is the result of two opposing forces. On the one hand, there is the infective factor, the virus; on the other, the resistance of the body. Each of these two factors may be modified by such variables as dosage or virulence of the infecting organism, or those influences which may temporarily lower the level of resistance, or hasten the development of antibodies. Whatever may be the variables involved, we know that whether or not disease develops depends on which of the two opposing factors is paramount. If, therefore, we are not completely successful in preventing the spread of the virus of rabies, can we not attack the problem through so raising the level of resistance of the dog population that the disease will not spread?

You are all familiar with the work carried on during the past fifteen years in an attempt to develop an effective antirabic vac-

cine for dogs. I do not need to review the work for you other than to point out that the methods have been improved both as to effectiveness and elimination of complications. I will not presume to give you an exact estimate of the level of resistance that can be expected from the single-dose method, for the evidence is still too incomplete to permit of a final or exact evaluation. Of this much we can, I think, be certain, *viz.*, that the annual injection of the vaccine at present available through the usual market channels does materially elevate the resistance of the dog against rabies. If it accomplishes nothing more than is embodied in this conservative estimate, it is still of tremendous value from the public health point of view.

We Speak Glibly of Immunity

You will note that I have purposely avoided the use of the word "immunity." I have preferred to do so because the word has an absolute connotation which to the public is deceptive. We never immunize a person against any disease. What is actually accomplished is to raise the resistance to the level necessary to protect the individual against the average dose of the infecting organism. It is not intended that the person given injections against typhoid should be able to drink a culture of typhoid bacilli, or that a child protected against diphtheria should be able to gargle with the Klebs-Loeffler bacillus. These are absurd situations against which we have not attempted to protect the individual. Yet, unfortunately, we have spoken glibly of immunity, when what we have actually meant has been a relative resistance sufficient to carry the individual through the average vicissitudes of life. In our enthusiasm, we have unconsciously led the public to expect too much and then have had to explain why a certain rare child developed diphtheria in spite of the injections.

In considering the application of such methods to rabies, can we not perhaps learn a lesson from our experience with diphtheria? At the outset, the work was carried on with toxin-antitoxin, a solution which will cause the subsequent Schick test to be negative in 65 to 90 per cent of those so treated. Subsequent research has produced improved antigens, toxoids which are from 95 to 98 per cent effective. Yet, in spite of the much lower efficacy of the earlier solutions, diphtheria has been effectively controlled. In this state, where until the past year virtually all the work has been carried on with toxin-antitoxin, diphtheria has been reduced to a figure less than one-tenth of its incidence ten years ago. This has been accomplished through a very wide-

spread use of toxin-antitoxin, accepting the fact that an occasional failure may appear to require explanation. In some communities, more than two-thirds of the children have received the injections, with the result that diphtheria has become a rare disease instead of the principal cause of child death. It has been estimated on good authority that if one-third of the children under five could be so protected, diphtheria would virtually disappear. Whether or not this be the correct figure, the principle of control of the disease, through raising the resistance of a substantial group of susceptibles, is sound.

The conclusions to be drawn from such experience can probably be transferred with little change to the problem of rabies. We do not know the exact level of protection established by the one-dose method of dog inoculation. Some say 65 per cent; other enthusiasts say 95 per cent. Probably neither is right, for so much depends on the strain and the dosage. We have no simple resistance test such as the Schick test for diphtheria. Figures based solely on laboratory tests are apt to give an underestimate, owing to the severity of the tests. It is, however, probably safe to say that our present methods give to the dog, for at least a year, a protection quite comparable to that conferred in the human against diphtheria through the use of toxin-antitoxin. From our own field observations so far made in this state we cannot be more definite. Further experience in practical use will give us, however, added information.

In the meantime, the health officer can well add to his antirabic armamentarium the weapon of dog inoculation. We do not need to wait for the development of a method which will protect every dog against an intracerebral inoculation of the most potent virus. As with diphtheria, progress will come as years pass. We are sure today that the method does increase the level of resistance, so it is fitting that its use should be further extended. But in so doing, it is well that we should avoid extravagant claims. There is no need for claiming for the method advantages that we do not know it to possess. Suffice it to say at the moment that it helps to protect the dog. It is far easier to teach the public that the method is more effective than we had realized than it is to disillusion a public whose hopes had been raised to dizzy heights. Nor should we be concerned with the fact that all dogs in the community are not reached. In diphtheria, virtual control is possible if less than half are protected, in spite of the fact that the existence of carriers may add to the menace for the unprotected. In rabies, we are not bothered, as far as we know,

by carriers. What figures are now available give us reason to believe that the analogy between the diseases is not too greatly at variance.

In concluding this discussion of rabies, as viewed by a health officer, I should like to be permitted for a moment to gaze into a crystal ball and see there the rabies control program that I hope we may some day achieve. If further experience with anti-rabic inoculation of dogs justifies our expectations, we might look forward to a much wider application on a self-sustaining basis. By this, I do not mean a compulsory vaccination of all dogs as a prerequisite to licensing; desirable as such might be, it seems unlikely that we would see that in the near future. We might, however, look forward to the time when, in return for the license fee, the dog-owner would be entitled to free inoculation, either in public clinics or in the veterinarian's office, the cost to be taken out of the dog license money. My neighbor who pays two dollars for his dog license should receive something more therefor than the privilege of having his dog dig up my garden. That is free, but the inoculations cost money and, unfortunately, the public will not avail itself of such methods unless obtaining them is made as simple and as cheap as possible. Such a method would appeal to the dog-owner, as the principal protection would be derived by his own cherished possession, the dog. What proportion of the dog population would be so reached, I do not know, but probably a sufficient proportion to make the spread of rabies slow and difficult. Under some circumstances inoculation might be offered as an alternative to permanent restraint, but this probably would not be necessary.

With such a reduction in rabies, we might look forward to such a low incidence of the disease that communities might be free of it for substantial periods, and the occasional case that developed might be dealt with by measures vastly more thorough than are possible today. Finally, and to the health officers most important of all, many of the people who today are forced to undergo anti-rabic treatment might be spared this ordeal. When that Utopian day shall arrive, the health officers may well feel that rabies is no longer a major problem. I hope to live to see that day.

Veracity Unquestioned

Fisherman: "I tell you it was that long. I never saw such a fish."

Friend (skeptical but polite): "I believe you."

CANINE DISTEMPER AND ITS CONTROL*

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In discussing this subject I do so appreciating fully that authorities are not in complete agreement on certain points. For the sake of brevity I have intentionally omitted a résumé of the voluminous literature on this subject and have endeavored to confine my remarks to information of practical interest to the practitioner. Neither will time permit the incorporation in this paper of the large amount of experimental data which my co-workers and I have accumulated and which support the statements to follow.

Canine distemper has been the subject of extensive research and discussion for the past ten years and will undoubtedly continue to be for a number of years. However, these investigations have resulted in marked progress, improving our knowledge of its pathology and control. It is hoped that further studies will result in a practical method of differentiating distemper of virus origin from that of bacterial origin and further improvement in the method of control.

Canine distemper, as recognized in practice, is an acute contagio-infectious disease characterized by coryza, an unusual but characteristic temperature curve, gastro-intestinal disturbances, pulmonary involvement and, in some cases, nervous and exanthematous manifestations. From a technical standpoint we are justified in speaking of canine distemper as a specific disease, but in practice it is rarely, if ever, observed in true, uncomplicated form; so that from a practical point of view the term, canine distemper, is applied to a group of disorders (coryza, bronchitis, pneumonia, gastro-intestinal enteritis, etc.) commonly associated and presenting a rather typical clinical picture. From an etiological standpoint, so-called "flu" of swine and influenza of humans are quite analogous, as it has been proven conclusively that a filtrable virus is the cause of the specific disease in each case, but the etiology of each as observed in practice is complex, being due to a virus and one or more bacterial organisms. In canine distemper there is no question but that the filtrable virus is the usual primary causative agent and that the following bacterial organisms should be considered as having etiological significance: *Alcaligenes bronchisepticus*, streptococci, *Salmonella paratyphosus*

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B., *Salmonella enteritidis*, and staphylococci. The occurrence and importance of the latter will vary with the seasons and localities and in general the importance of the organisms is in the order given. Since we have no practical diagnostic test for virus infection, and clinical differentiation of virus from non-virus infection is usually impossible in practice, it is necessary to consider the whole syndrome as canine distemper.

We may summarize our knowledge of the etiology as follows:

1. Canine distemper as observed in practice is a mixed infection in which the filtrable virus is usually the primary factor and bacterial organisms are responsible for the principal symptoms and lesions, the severity of the disease, and the high mortality.
2. Only one distinct strain of virus has been demonstrated conclusively; however, variations of virulence occur.
3. The virus is infectious for ferrets, foxes, and other fur-bearing animals; however, there is evidence that distinct strains are responsible for distemper as it occurs naturally in such animals.
4. The bacterial pathogens (*A. bronchisepticus*, streptococci, *S. paratyphosus* B., *S. enteritidis* and staphylococci) are the important secondary invaders and in some instances may act independently of the virus as the primary cause of a distemper-like syndrome.

Distemper is most commonly observed in puppies from six weeks to nine months of age. Younger puppies may occasionally become infected, particularly if from non-immune mothers. Older dogs are susceptible if they have never contracted the infection. Evidence indicates that virus infection confers a lasting immunity against true distemper; however, such dogs may contract a distemper-like disease of bacterial origin. Fortunately, the prevention of virus infection removes the prevailing predisposing factor to bacterial infection. It is doubtful if any given breed is more resistant than another but certain strains are undoubtedly more resistant. Diet, particularly the vitamin and mineral elements, without doubt, influence susceptibility to a marked degree; however, the claim that the disease may be satisfactorily prevented by a special diet, has never been substantiated.

The period of incubation varies with the virulence of the virus, method of exposure, and resistance of the animal. Artificial inoculation causes a temperature elevation in three to six days (average four days), while from natural exposure the period ranges from four to ten days, or longer, with an average of about one week.

Symptoms

The symptoms of canine distemper are variable, as would be expected in a disease of such complex etiology. A history of inappetence, shivering, sluggishness, sneezing or coughing, nasal or eye discharge and abnormal bowel movements are very suggestive. The actual onset of the infection is characterized by an initial temperature elevation of 103 to 105° F., or higher, which continues for 24 to 48 hours and then returns to normal. At this stage visible symptoms, other than possibly slight inappetence, shivering, sluggishness, and frequently, vomition, are not observed. Within two or three days a second rise of temperature, usually not so high as the first, occurs and continues remittently throughout the course of the disease. Accompanying the second elevation of temperature, marked visible symptoms appear. Watery eye and nasal discharges are observed, slight at first but increasing, becoming thicker and forming crusts about the inner canthus of the eyes and around the nostrils. The appetite is capricious or lost. Sniffling or a hacking cough is common; severe, incessant cough is indicative of pulmonary involvement. Constipation may be evident at onset but a tendency to diarrhea is soon observed and in severe cases is profuse with evidence of a hemorrhagic enteritis. The character of the pulmonary and intestinal symptoms and the condition of flesh indicate the severity of the disease. The symptoms vary with the character, site and virulence of the bacterial infection. Pneumonia, hemorrhagic enteritis, ophthalmia, nervous manifestations and pustular skin eruptions are common in the advanced stage of severe attacks. Deaths are usually the result of pneumonia, enteritis, or, more frequently, a combination of the two.

The nervous symptoms, especially the choreic type, are seldom in evidence before the third week. Evidence indicates that these symptoms are the result of the infection, be it virus, bacterial or their exogenous products, involving the central nervous system. Microscopic, cultural and inoculation examinations in such cases have failed to incriminate a specific organism. *A. bronchisepticus*, streptococci, *S. paratyphosus* B., or *S. enteritidis* may be isolated frequently from the central nervous system.

Course and Diagnosis

Acute cases usually recover or die in two to four weeks while chronic or seriously complicated cases may run a course of four to twelve weeks. Our records show that the average course of the disease, whether it ends in recovery or death, is 29 days. A

high temperature in the advanced stage, marked emaciation, great exhaustion, shallow respiration, nervous symptoms and coma are unfavorable signs.

The diagnosis of distemper in the early stage is frequently very difficult, particularly if there is no history of exposure. A history of exposure and the detection of symptoms suggestive of distemper, particularly in dogs under one year of age, are sufficient to justify a tentative diagnosis of distemper or, at least, such cases should be treated as distemper until proven otherwise. It is frequently impossible to differentiate distemper of virus origin from catarrhal disorders (bronchitis, pneumonia, gastroenteritis, conjunctivitis, laryngitis, etc.) of bacterial origin. The characteristic temperature curve at the onset is of valuable diagnostic importance, but unfortunately the practitioner seldom has an opportunity to observe it.

Practitioners having laboratory facilities may find the blood picture valuable in arriving at a diagnosis and subsequent prognosis. For that reason the following data from our research files may prove of interest:

Normal Physiological Data

Average normal temperature—based on 200 temperatures of 41 dogs				101.48
Average normal white cell count—based on 73 examinations of 41 dogs				11,599
Average normal red cell count—based on 32 examinations of 32 dogs				6,541,250
Average hemoglobin percentage—based on 41 examinations of 41 dogs				98.1
Average normal Schilling differential—based on 73 examinations:				
Basophiles	0.16			
Eosinophiles	1.46			
Myelocytes	2.29	Immature	23.29	Neutrophiles
Juveniles	4.73			
Stabs	16.27			
Segments	45.38	Mature	45.38	
Lymphocytes	28.41			
Monocytes	1.3			
Nuclear index	1.95			

We find that one is not justified in basing a diagnosis on the blood picture (Schilling method) alone; however, when considered in conjunction with the history and symptoms it is of considerable diagnostic and prognostic value. The complex nature of canine distemper, as related to its etiology, renders interpretation difficult and likely to be misleading. In general, one may consider the following changes in the blood picture as being characteristic of canine distemper as it occurs in practice.

1. A leucopenia occurs quite consistently with, or just following, the initial temperature rise and visible symptoms, the

white blood cell count falling within a range of 5 to 10 thousand, an average of about 8,000, with no significant change in the nuclear index.

2. Coexistent with the second elevation of temperature and the onset of bacterial infection, the white blood cell count rises and is accompanied by a drop in the nuclear index and a decrease in the number of lymphocytes. With severe complications the white cell count may reach 40 to 80 thousand just before death, with a nuclear index of 0.5 or even less. The lymphocyte count may drop from normal, 25 to 30 per cent, to 5 per cent or less in cases ending fatally. The average high white cell count reached at the end of two weeks of visible sickness is about 20,000.

3. Although a leucopenia is characteristic of virus infection, it is not always present in cases known to be so affected. Furthermore, one must take into consideration the possible influence of parasitism and other infections in judging the significance of a blood cell count.

4. A marked drop in the nuclear index and lymphocyte count justifies a doubtful or unfavorable prognosis.

Lesions

The lesions observed on autopsy other than enlargement of the spleen and lymph-glands and occasionally scattered petechiae, are due to bacterial infection, therefore, varying with the site of infection, organisms involved and the course of the disease. In subacute or chronic cases, marked lesions will be observed in either the respiratory or gastro-intestinal tracts, or in both. In the respiratory tract, edema, congestion, hemorrhagic spots and a varying degree of pneumonia may be expected. The gastro-intestinal tract may show an enteritis varying from a catarrhal to a hemorrhagic or ulcerative type. Inflammatory exudates in the peritoneal cavities are common. Differentiation of virus from non-virus involvement is not possible by character of lesions, particularly because in the advanced stage virus is no longer an active factor.

Control

After considering the complex nature of distemper, its etiological factors and the pathological conditions possibly resulting from such infections, is it any wonder difficulty is encountered in prevention and treatment? Personally, I think the progress made during the past few years is quite remarkable even though

no one given product or method of immunization has as yet proven universally satisfactory. If we were dealing with a true entity, distemper caused by filtrable virus alone, the problem would be simple but under the circustances I doubt if it will be possible to perfect a simple method of immunization which will prove universally satisfactory. There is a tendency to rely too much on what we believe or hope is a specific, overlooking factors which lower resistance and thus influence the results obtained. Therefore, it is essential that we keep in mind sanitation, diet, parasitism, proper care, etc., as factors influencing susceptibility to and immunization against distemper. It is highly probable that diet, as it pertains to mineral balance and vitamin content, particularly the latter, has a marked influence on predisposition to infection and the degree of immunity obtained by vaccination. Vitamins A and D are of particular importance.

Biologics

Beyond question, the development of homologous anti-canine distemper serum has resulted in the most effective product now available for the control of distemper. Marked improvement of the product has been effected by increased hyperimmunization of the serum-producing dogs. The original procedure was to give one hyperimmunizing dose and then bleed; now we administer repeated doses of virus over a period of 60 days before the initial bleeding and, also, between bleedings. In addition to virus antigen, bacterial antigen is used so as to result in a highly potent serum, active against both virus and bacterial organisms. Although it is not possible to titrate accurately the potency of the serum, it is possible to evaluate its strength by testing in a manner similar to that used for anti-hog cholera serum. Laidlaw and Dunkin attempted to develop a laboratory method of standardization and gave a tentative report on the use of the complement-fixation test; however, to date this method has not proven satisfactory. This test, like the agglutination test, indicates the presence of complement-fixing and agglutinating antibodies, respectively, but this does not necessarily indicate that protective antibodies are present in equal concentration. The fact that accurate titrations of serum, other than antitoxins, are not yet possible, that this is particularly true of an analogous product, anti-hog cholera serum, indicates that we must depend on an animal protection test for determining the potency of anti-canine distemper serum.

Recently, a practical method of concentrating homologous serum has been perfected, whereby the inert fractions are re-

moved and the protective fractions retained, reducing the volume and dosage to one-fifth that of the unconcentrated. The concentrated product is more readily absorbed and utilized by the patient as a result of its purification and reduced dosage. There is no appreciable loss of total antisubstance from concentration and such loss is more than compensated for by the increase in effectiveness resulting from purification. Concentrated serum is especially desirable for use on very small or delicate breeds of dogs where a dose of large volume is undesirable. Field and laboratory tests have shown conclusively that either product will protect against the usual dose (10 mg) of virulent, desiccated spleen virus administered simultaneously or even when the virus is given 24 to 48 hours before the serum.

Homologous serum, concentrated and unconcentrated, has a high bacterial antibody content in addition to its virucidal properties, rendering it effective for passive immunization and curative treatment of distemper in any form or stage. Being a homologous product, it may be administered in liberal dosage without danger of shock or anaphylaxis. Passive immunity is relatively short, usually not more than ten to 14 days. Curative effect is usually observed after one or two doses when used in the early stage of the disease. For prophylaxis the usual dose is ten to 30 cc of unconcentrated or two to four cc of concentrated serum, given subcutaneously or intramuscularly. For treatment, 10 to 30 cc of unconcentrated or four to eight cc of concentrated for dogs of 20 pounds or under should be given subcutaneously or preferably intravenously. In larger dogs the dose should be increased, giving 1 cc of unconcentrated serum or a corresponding amount of concentrated for each pound of body weight. The initial dose should be large, particularly in the early stage of the disease, repeating at 24- to 48-hour intervals until the temperature is normal and the patient is eating. The results are more satisfactory and the treatment is more economical if the initial dose is large, as this frequently reduces the number and size of doses necessary to complete the treatment. Since the serum has a high bacterial antibody content, it may be used satisfactorily in treating advanced cases. However, it is more economical to use anti-mixed infection serum or mixed bacterin (canine) for that purpose.

Canine distemper tissue vaccine is a 20 per cent suspension of spleen tissue virus in which the virus has been killed with formalin. Although almost any preservative will destroy the virus rapidly, formalin is chosen because it has the property of increas-

ing the antigenic value of the product. Tissue vaccine confers a relative degree of active immunity which becomes effective in about ten days following injection. Two doses are recommended, allowing an interval of ten to 14 days between doses. In the majority of cases a satisfactory immunity is afforded against virus infection, but no immunity to bacterial infection is obtained.

Virogen is a product which is identical to regular tissue vaccine except that in addition to 20 per cent spleen tissue virus it contains bacterial antigens equivalent to at least one dose of bacterin. This product confers just as high a degree of immunity against the virus infection and has the additional advantage of immunizing against the bacterial infection. Field results have shown the superiority of this product over the former, particularly in sections where bacterial infections are of outstanding prevalence. Virogen and tissue vaccine are more readily absorbed when injected intramuscularly in the side of the neck. On account of the tissue content of these products subcutaneous injection results in slow absorption, particularly if the injection is superficial and not distributed.

Canine distemper virus is one of the most delicate viruses known and for that reason exceedingly careful attention must be given to its preparation and distribution. To date, the only satisfactory source of virus for preparing commercial virus is the spleen of artificially infected animals. Although the virus is present in the blood and other tissues, it is found more uniformly and in higher concentration in the spleen. No method has been found whereby the virus may be preserved satisfactorily in liquid form and it is very doubtful if such a method is possible. It is an accepted fact that viruses in general are best preserved by drying and this is especially true of viruses which have a tendency to be of low resistance such as is distemper virus. Spleen tissue virus, properly desiccated and refrigerated, may retain satisfactory virulence for 90 days or even longer under properly controlled conditions. The virulence and safety of each lot of virus are determined before releasing and as an added precaution the return date is limited to 30 days from date of preparation.

Virus is for use only on healthy dogs in conjunction with canine distemper tissue vaccine, virogen or anti-canine distemper serum (homologous). The recognized standard dose is 10 mg, suspended in suitable amount of diluent, which may be administered subcutaneously, intradermally or intramuscularly.

Anti-mixed infection serum and mixed bacterin (canine) are still useful and economical products for the control of canine dis-

temper. The serum is recommended for the prevention of those bacterial infections simulating canine distemper and the treatment of advanced or chronic cases of distemper in which the virus is no longer an active factor. Mixed bacterin is indicated for conferring an active immunity against the common bacterial infections which occur independently of the virus or as complications. In many localities bacterin is used in conjunction with the vaccine, vaccine-virus and serum-virus methods, to obtain a more complete immunity. Bacterin is useful in treating chronic cases or those where conditions do not justify the expense of using serum.

Prophylaxis

1. Passive immunity to canine distemper is obtained immediately on administration of anti-canine distemper serum (homologous), concentrated or unconcentrated. The duration of this immunity is relatively short, an average of about ten days. Ten to 30 cc of unconcentrated or two to four cc of concentrated serum is a satisfactory dosage. The dosage should be varied somewhat with the size of the dog and the severity of the exposure anticipated. Passive immunization is indicated wherever immediate protection is required and where the circumstances, such as previous or probable exposure, debilitated condition of the patient, suckling puppies, surgical operations, etc., do not permit active immunization.

2. Active immunization may be accomplished in various ways but in every case the action of virus, through inoculation or exposure, is required. At this time it is impossible to say that any given method is universally superior, as distemper varies in character and severity in different localities. For that reason it is recommended that where a given method is giving satisfaction, the practitioner should proceed with caution in making any change.

The vaccine and vaccine-virus procedures have proven reasonably effective in most instances. Two doses of canine distemper tissue vaccine, or preferably virogen, with an interval of ten to 14 days between doses, confer a high relative degree of immunity which is sufficient to protect against virus infection of average virulence, virogen being superior to regular vaccine for the reason given previously. The administration of virus 14 days subsequent to the last dose of vaccine results in a higher degree of immunity and is recommended where it has been found that the virulence of the existing infection is high. A single dose of vaccine followed by virus has not proven sufficiently safe or effective to justify

general use. The principal objections to the vaccine and vaccine-virus methods are the number of doses required and the fact that the patient is susceptible during the vaccinating period; in fact, the animal is more susceptible after receiving the first dose of vaccine since the resistance is lowered temporarily, and the full immunizing effect is not obtained until about the tenth day following its use. In order to avoid possible infection from exposure during this period, it is necessary to give a dose of serum simultaneously with the first dose of vaccine.

The serum-virus method, although comparatively new in this country, has been used quite extensively in England and has been the subject of a large amount of field and laboratory experimental work, especially during the last two years. The development of a more potent serum and stable virus has been responsible for the recent progress made with this method of immunization. Extensive laboratory and field vaccination tests, the latter covering practically all sections of the United States and involving several thousand cases, have proven conclusively that a potent serum will protect against the usual dose (10 mg), or even more, of virulent virus even when the latter is given as long as 72 hours before giving the serum. The simultaneous use of serum and virus confers a high degree of active immunity which to date has proven to be highly satisfactory. The indications are that the method results in permanent immunity against the filtrable virus in more than 90 per cent of the cases. The method has the advantage of requiring only one vaccination treatment, conferring immediate immunity, being suitable for dogs of any age, and that it may be used regardless of a knowledge of previous exposure, provided the dog is in good physical condition at the time of vaccination. Of course dogs known to be in the incubation period may in some instances require an increased dose of serum, or more than one dose. Suckling puppies should not be treated before one month of age and even then serum alone is probably best, deferring serum-virus treatment until after weaning.

Knowing that within limitations the degree of immunity is in direct proportion to the degree of virus reaction, it has been determined by laboratory tests that a somewhat higher degree of immunity is obtained by giving the virus 24 to 48 hours before giving the serum, thus permitting the virus to incubate in the body and excite more cellular reaction before being counteracted by the serum. This method should be used in treating suckling puppies after the first month of age (they usually possess considerable inherent immunity) and may be used to advantage in all

dogs which are known not to have been exposed, are in healthy condition, and which can be kept subject to the practitioner's observation during the reaction period (ten days). This precaution is necessary to avoid the possibility of excessive reaction resulting from exposure to the use of virus. A temporary temperature elevation at four to seven days after the virus injection is desirable and no cause for worry. Should excessive reaction, visible symptoms or continuous high temperature be noted, a large dose of serum should be given promptly to prevent further developments; however, to date, we have not experienced excessive reactions.

The dosage of serum for the serum-virus method is ten to 20 cc of unconcentrated serum (20 cc for dogs of 15 pounds, or over) or 4 cc (2 to 4 cc for suckling puppies) of concentrated serum, given simultaneously with 10 mg of virus, at different sites, or 24 to 48 hours after giving virus. These procedures confer an active immunity against the virus, but only a passive immunity against the common bacterial invaders. To activate the immunity against the latter it is necessary to use mixed bacterin (canine) which is advisable in localities where it is known that highly virulent bacterial infections are common. An accurate balance of the serum and virus dosage is not essential to active immunization. As in the simultaneous treatment for hog cholera, it has been shown conclusively that a reasonable excess of serum does not lower the degree of active immunity to any appreciable degree. In order for any simultaneous vaccination, using serum and virus, to be safe and practical, a slight excess of serum must be used to take care of possible variations in the virulence of the virus and, also, variations in the susceptibility of the patients.

SUMMARY

1. Canine distemper as it occurs in the United States is a complex disease. The filtrable virus is the cause of true, uncomplicated distemper and usually the primary exciting factor of distemper as commonly met with in practice.

2. Bacterial pathogens are responsible for the principal symptoms, lesions, and mortality and in some instances, alone, are responsible for so-called canine distemper.

3. The control of canine distemper is largely dependent on the use of biologics, which is rendered difficult by the complex etiology, but fortunately immunization against the virus overcomes the principal predisposing factor (virus infection) to so-called distemper of bacterial origin.

4. An attack of distemper, having virus as the primary factor, results in a solid, permanent immunity against virus infection. Administration of vaccine or virogen, two doses, results in a marked, relative immunity against virus infection which may be further activated by the injection of virus. In addition, virogen confers a relative immunity against the common bacterial invaders.

The serum-virus method confers a solid active immunity against the virus and a passive immunity against the bacterial invaders. The advantages of this method are: fewer doses, immediate protection, fewer contra-indications, and the results to date indicate that a higher degree of active immunity is obtained more universally than by any other method. Results to date indicate that the simultaneous method gives a permanent immunity in at least 90 per cent of the cases and that the administration of virus 24 to 48 hours before giving the serum is still more effective. It is the writer's opinion that the serum-virus method will gradually supersede all other methods of vaccination.

Court Decision Favors Doctor Sutton

The Georgia Supreme Court, in a decision rendered December 17, 1934, held that Dr. J. M. Sutton was illegally removed as State Veterinarian of Georgia when his name was stricken from the salary list of the Department of Agriculture and he was locked out of his office. The decision marked another chapter in the long-drawn-out legal battle between Dr. Sutton and Governor Eugene Talmadge whose differences with the State Veterinarian began while Talmadge was Commissioner of Agriculture.

The decision of the Supreme Court held that Judge Hutcheson, of the Dekalb County Superior Court, had erred in granting a demurrer to a group of officials who had been made defendants in an injunction suit brought by Dr. Sutton to have the state officers restrained from interfering with his work as State Veterinarian and ordering them to restore his name to the state pay-roll.

It is reported that the State will file a motion for a rehearing of the case before the Supreme Court. Unless the ruling is reversed, the legal obstacles are removed and Dr. Sutton will be enabled to collect the salary of $4,200 due him for his work as State Veterinarian during the past year.

OVARIAN CYST IN A CAT*

By WM. R. KERMEN, *Los Angeles, Calif.*

Ovarian cysts are common in cats, but during the past few years no enormous ones have been reported. Occasionally, in the past, enormous fibroids and other tumors of the female genital organs were seen, but they have been detected and removed before they reached such an enormous stage.

REPORT OF CASE

History: Spayed female cat, 17 years old, was first seen a month ago. The owner complained that the cat could not urinate, and directed attention to the enormous size of the abdomen. The cat showed no signs of nervous excitement or epileptiform convulsions. Due to her great size, she moved about sluggishly, with her abdomen dragging on the ground. The owner had noticed a gradual increase in size over a period of years, but this was the first time that she had had a veterinarian look at the cat.

Examination: The patient weighed 17.8 pounds. The temperature was 99.2° F; the respiratory rate was 20; moist râles were heard at the base of each lung. The abdomen was enormously distended and was flat on percussion. On palpation, a large distended organ which resembled the bladder was felt. A pre-operative diagnosis of calculi in the bladder was made, but it would have been ruled out if the history was correct. Ascites was considered but was ruled out by the absence of edema of the extremities and no shifting of the fluid on changing positions. Cystic formation in the ovary was not even thought of, as the cat was supposed to have been spayed. She showed no nervous symptoms, no prolonged estrum and no epileptiform convulsions.

Operation: Under local anesthesia, a trocar was passed through the abdominal wall. One and four-tenths liters of dark fluid was gradually withdrawn. The patient succumbed to the operation two hours later.

*Received for publication, November 13, 1934.

Autopsy: On autopsy, we found a cyst that originated from what remained of the left ovary. We found that the trocar had been passed into the cyst instead of the bladder. The bladder was normal, but owing to the enormous size of the ovarian cyst, it was impossible for the cat to urinate. The uterus was greatly enlarged, and both horns contained multiple fibroids. The omentum also contained multiple fibroids, which proved to be scirrhous adenocarcinoma on microscopical examination.

Comment

Cystic formation in the ovaries is of frequent occurrence and perhaps much more common in cats than in any other animal. This case is interesting because of the enormous size of the cyst, originating from only part of the ovarian tissue, and the multiple tumors throughout the uterus and omentum.

October Sets Record in Tuberculosis Eradication

During the month of October, 1934, a new record was established in connection with bovine tuberculosis eradication, when 1,805,202 cattle were tested in the 48 states. This number was the largest for any month since systematic work was started in 1917. Of the large number of cattle tested, 49,932 reacted and were designated for removal and slaughter, another all-time monthly record.

Commenting on the relatively high percentage of reactors (2.8 per cent), Dr. A. E. Wight, Chief of the Tuberculosis Eradication Division of the U. S. Bureau of Animal Industry, explained that this was the result of the concentration of testing in areas where tuberculosis infection was high. The largest number of cattle tested in any one state during October was 260,791, in Minnesota. Other states reporting large figures were New York, 209,078; Iowa, 186,871; Missouri, 119,691, and California, 53,570.

The increase in activities was made possible by emergency funds made available by the federal government for both operating expenses and indemnity payments. Several of the states have been taking advantage of the opportunity afforded for pushing the work to completion at a much earlier date than otherwise would have been possible.

A man's best things are nearest him,
Lie close about his feet.—Lord Houghton.

LEHRGANG DER HISTOPATHOLOGIE FÜR STUDIERENDE UND TIERÄRZTE. (Textbook of Histopathology for Students and Veterinarians.) Oskar Seifried, Professor des Institutes für Tierpathologie der Universität München. 195 pages and 142 illustrations. Julius Springer, Berlin, 1934.

Books on veterinary histopathology are quite rare. Doctor Seifried from his work in this field and from his familiarity with this subject has written a very practicable and usable text. The great need in this field will make this text appeal to those interested in histopathology.

The author considers in the first part of his book the regressive changes from a general pathological standpoint. He then discusses the progressive cell and tissue changes including regeneration, hypertrophy, hyperplasia, metaplasia and organization together with the histopathological changes found in the various kinds of tumors.

The second part of the book is devoted to special pathology. The various organs and systems of organs including a section on blood and blood-forming organs are discussed. Particularly interesting in this discussion are the references in connection with the organs and organ systems to the special forms of diseases affecting such tissues. The author has drawn from his experience in Germany and in the United States with such diseases as avitaminosis, rhinitis, infectious laryngotracheitis and fowl-pox, and treats of these diseases when discussing the respiratory tract. He has also very well illustrated the changes by photomicrographs and some drawings. Dr. Seifried's special studies on the histopathological changes in hog cholera make this phase of his discussions particularly interesting.

The third part of the book is devoted to the histopathological changes occurring with virus diseases, bacterial diseases, parasitic diseases, deficiency diseases, toxic diseases and the results of thermal, electrical and mechanical disturbances.

The book is by no means complete in all details but is of greatest interest because Dr. Seifried has drawn from his wide ex-

perience and has given the work a more or less personal touch. The photomicrographs are excellent as well as some drawings and graphic illustrations that the author has made. The book warrants wide distribution and is a very distinct contribution to the meager sources of veterinary histopathology.

E. L. S.

A VETERINARY HISTORY OF NORTH CAROLINA. William Moore, L. J. Faulhaber and J. H. Brown. 54 pages. North Carolina State Veterinary Medical Association, Tarboro, N. C., 1934.

At the 1934 meeting of the North Carolina State Veterinary Medical Association, held July 10-11, a motion prevailed authorizing the Secretary to have prepared and published a history of the Association and the veterinary profession in North Carolina. With unusual promptness in such matters, a committee consisting of Drs. William Moore, L. J. Faulhaber and J. H. Brown set about their task, and the date of the completion of their work is shown as October 1, 1934.

The veterinarians of North Carolina have reason to be proud of the history of the profession in the state, and it is to be hoped that the profession in other states will appreciate the value of recording veterinary history while it is possible to do this accurately. With a frequency that is sometimes disquieting, the pioneers of the profession are passing away and many of these men possess information concerning the early history of the profession that is not recorded in any form, shape or manner. Veterinarians have been slow to record in permanent form many facts and, as time passes, the task of collecting and arranging this information becomes increasingly difficult. The only other publication in the form of a history of a state association is the booklet recording the transactions of the Virginia State Veterinary Medical Association, compiled and published by Dr. George C. Faville, of Richmond, Va., about four years ago.

The history of the veterinary profession in North Carolina contains many names that are connected with the veterinary history of the United States. Dr. Cooper Curtice, now of Virginia, Dr. Tait Butler, now of Tennessee, Dr. E. M. Nighbert, now of Florida, and the late Dr. J. A. Kiernan are just a few who, at various times, were active in veterinary work in North Carolina.

It is surprising how much information has been collected and arranged to tell the story of the veterinary profession in North Carolina. The history includes chapters relating to the office of the State Veterinarian; the U. S. Bureau of Animal Industry; a

list of all graduate veterinarians who have worked in North Carolina; a section on contagious and infectious diseases, including poisonous plants and poisonous snakes; the Constitution and By-laws of the North Carolina State Veterinary Medical Association, with a copy of the act of the General Assembly incorporating that body. Then follows a section containing reports of the annual meetings of the Association from 1907 to date. In this connection, it is pertinent to remark that reports of the first to fifth annual meetings appear to be lost. The history is concluded with lists of active and honorary members of the Association; a list of members who have died; a list of past officers, as well as a list of the annual meetings. A short chapter is devoted to the North Carolina Board of Veterinary Medical Examiners, with personnel and officers of the Board from 1907 to date; a list of the A. V. M. A. Resident Secretaries for North Carolina; a list of A. V. M. A. members located in the state, and a concluding paragraph is devoted to the Ladies' Auxiliary of the North Carolina State Veterinary Medical Association.

It is to be hoped that other state associations will take steps to have their histories compiled and published before the material is lost or too badly scattered, and while we have with us men who can be of material assistance in doing the job.

HANDBUCH DER CHEMOTHERAPIE. ZWEITER TEIL, METALLDERIVATE. (Manual of Chemotherapy. Part II, Metallic Derivatives.) Victor Fischl and Hans Schlossberger. xi+540 pages. Fischer's Medical Bookbinders, Leipzig, 1934. Price, M. 55.

The first volume, a review of which appeared in the JOURNAL,* dealt with the chemical constitution and therapeutic use of a large number of metal-free organic compounds. The second volume deals with metallic derivatives. The following metals are discussed: Arsenic, antimony, bismuth, iodine, copper, silver, gold and mercury. One chapter is devoted to rare metals, such as vanadium, platinum, iridium and osmium. There are reviewed, also, such elements as fluorine, magnesium, aluminum, phosphorus and sulfur. Detailed lists of references are given.

In the two volumes of this manual, the authors have undertaken to publish a book that covers the field of chemical therapy in all its aspects. Such a work has not been attempted before, and an effort has been made to collect all available data on chemical therapy and include them in one book. This is the first work

*Jour. A. V. M. A., lxxxiii (1933), n. s. 36 (1), p. 112.

of its kind, according to the authors, due to the fact that chemical therapy is linked with many branches of natural science and medicine, and the subject has been treated, therefore, in a variety of scientific periodicals and publications.

DISEASES OF POULTRY: THEIR PREVENTION AND TREATMENT. H. P. Bayon. 155 pages, 37 illustrations. The Feathered World, Ltd., London, 1934. Cloth, 3/10 postpaid.

The book is offered as a practical hygienic guide for the poultry-keeper. The author has made an attempt to place emphasis upon the diagnosis of specific diseases, their treatment and prevention. Considerable pathology is given and it sometimes appears to be rather technical for the layman. Considerable discussion is given to the subject of nutrition—rational feeding, food deficiencies and excesses—which occasionally also becomes rather technical. A chapter each deals with the subjects of physiology, housing, and breeding from a health standpoint. Subsequent chapters discuss diseases caused by bacteria, moulds and viruses, parasitic infestations, mechanical injuries and poisons. The last two chapters of the book are devoted to general hygienic measures and how to perform the postmortem examination of a fowl. The subject matter is nicely arranged and easily read. Even through disease prevention is stressed, one might find opportunity for rather severe criticism of the treatments outlined for certain specific diseases; many such treatments are impractical from the standpoint of the practical poultryman.

M. W. E.

POULTRY AILMENTS. W. P. Blount. 306 pages, 30 illustrations. Poultry World, Ltd., London, 1934. Cloth, 5/— net.

The main object of this book is to present to the intelligent poultry-keeper facts which will help him to understand and interpret more clearly postmortem reports. Veterinarians, agricultural and poultry students should also find the book of much value. The subject matter is divided into two major parts, avian physiology and diseases. Fully one-half of the book is devoted to a critical discussion of avian physiology. The physiology of every major system, even the endocrine system, is discussed. The various diseases are arranged alphabetically and are discussed in considerable detail. One chapter is devoted to the transmission of disease among birds and is quite out of the

ordinary, as it also discusses infections that are transmissible to man. Another chapter deals with disinfection. The last chapter is devoted to a short discussion of research work. In general, the book appears even too technical for the intelligent poultry-keeper, although the author has made a splendid effort to make it understandable and has even included a glossary of the more technical terms.

M. W. E.

PUBLICATIONS RECEIVED

Washington Monthly Bulletin, State College of. Announcement for 1934. (Pullman, Wash., 1934. pp. 309.)

Nevada State Board Stock Commissioners, Biennial Report from July 1, 1932, to June 30, 1934. (Carson City, Nev., 1934. pp. 34.)

Nevada State Rabies Commission, Biennial Report from July 1, 1932, to June 30, 1934. (Carson City, Nev., 1934. pp. 8.)

Enzoötic Botulism Amongst Wild Birds. E. Murray Pullar. Reprint from *Austral. Vet. Jour.*, x (1934), 4, pp. 128-135. Illus.

The Incidence in Great Britain of *Br. Abortus* Infection in Various Domestic Animals as Indicated by the Agglutination Test. F. W. Priestly. Reprint from *Jour. Comp. Path. & Therap.*, xlvii (1934), 3, pp. 181-189.

Non-Pathogenic Haemolytic Streptococci Occurring in Milk. F. C. Minett and A. W. Stableforth. Reprint from *Jour. Dairy Res.*, v (1934), 3, pp. 223-233. Illus.

The Precipitin Test as a Means of Diagnosis of Tuberculosis in Cattle. Janet McCarter, Walter Wisnicky and E. G. Hastings. *Amer. Rev. Tuber.*, xxx (1934), 5, pp. 584-587.

Our Changing Agriculture Served by Science. Fiftieth Annual Report of the Director, Agricultural Experiment Station, University of Wisconsin, Madison, 1932-1933. (Bul. 428, Wis. Agr. Exp. Sta., June, 1934. pp. 128. Illus.)

International Agricultural Directory. J. W. Pincus. (Wm. Grant Wilson, Cambridge, Mass., 1934.)

Further Studies of Diseases Affecting Moose. R. Fenstermacher. (Bul. 308. Minn. Agr. Exp. Sta., Sept., 1934. pp. 26. Illus.)

Diseases Affecting Moose. R. Fenstermacher. Reprint from *Vet. Alumni Quar.*, xxii (1934), 3, pp. 81-94.

History of U. of P. Veterinary School

Alumni of the School of Veterinary Medicine, of the University of Pennsylvania, are being asked for contributions to assure the publication of a history of the School, which will be a permanent marker of the half-century milestone which the School attained last year. According to the *University of Pennsylvania Bulletin*, 100 contributions of $2 each are needed before work on the history can go forward.

CERTAIN PROPERTIES OF THE VIRUS OF EQUINE ENCEPHALOMYELITIS. Beatrice F. Howitt. Jour. Inf. Dis., lv (1934), 2 p. 138.

The virus of equine encephalomyelitis (California strain) passes without difficulty through the ordinary filter candles and has been shown by Krueger to measure approximately 500 millimicrons. The virus filtered through a Seitz disk may be diluted 1:900 to 1:2,000 and remain viable, while the unfiltered suspension withstands a dilution of from 1:10,000 to 1:50,000. Brain tissue may be preserved in 50 per cent glycerin for one year but loses virulence more readily if kept in a dried condition. Twenty per cent suspensions of virus withstand freezing for 13 months. The filtered material is more easily destroyed. A 1:10 saline dilution of the filtered virus is more stable than a similar dilution in water and remains viable for at least one month in the icebox, two weeks at room temperature and 20 hours at 37°C.

Active virus in the blood-serum is not destroyed within from 15 to 20 minutes at 56°C., and when unheated retains viability if kept on ice for at least seven months. The filtered virus is more sensitive to strongly acid than to strongly alkaline reactions, losing viability below pH 5.5, but retaining it at pH 9.2. Suspensions of the virus were resistant to lysol, merthiolate, corrosive mercuric chloride and chlorinated lime in the concentrations used. They were especially resistant to phenol in concentrations below 3 per cent, but were destroyed by formaldehyde, chlorazene, and hydrogen dioxide in amounts which were ineffective when used for phenol. The virus was not precipitated by acetone in the dilutions employed. The virus may be recovered from the testes and the ovaries of experimentally infected guinea pigs killed either during the febrile period or after prostration. The urine was never found infectious and the feces were found so but once when the material was removed at the height of the fever. No virus was recovered from the ovary, udder, milk, suprarenal glands or filtered salvia of an experimentally infected mare. There was no difference in the neutralizing power of an immune serum, whether it remained in contact with the blood

clot for nine days, 17 days, 60 days, or 165 days, respectively; nor was the presence or absence of fresh complement of any significance in the test. Immune serum heated to 56°C. for 15 minutes was as effective as that containing complement.

INSTITUTIONAL AND OCCUPATIONAL UNDULANT FEVER IN COLORADO. Ivan C. Hall and Robert Learmonth. Jour. Inf. Dis., lv (1934), 2, p. 184.

Whenever possible tests on both blood-serum and milk-serum should be made to detect agglutinins for *Bact. melitensis* (or *Bact. abortus*) in dairy cattle. Tests of blood-serum are superior to tests of milk-serum. Undulant fever is transmitted most commonly, first, through handling infected meat in slaughterhouses, and second, through drinking raw milk from infected dairy cattle. All the milk should be pasteurized to protect the inmates of institutions against infection with undulant fever from cows harboring abortion disease. Meat inspectors run a much greater risk of contracting undulant fever than do employes in anti-hog cholera serum plants.

STUDIES ON A HERD INFECTED WITH BRUCELLA ABORTUS. II. Incidence of milk contamination in a vaccinated herd. Dorothy W. Caldwell, Neil J. Parker and E. M. Medlar. Jour. Inf. Dis., lv (1934), 2, p. 235.

Twenty-three and six-tenths per cent of the cows which showed agglutinins for *Br. abortus* in their blood-serum were discharging the organisms in their milk. Seventy-two per cent of these cows were vaccinated and the remaining 28 per cent were unvaccinated. Many of these cows were discharging the organisms so regularly and in such numbers that infection of the udder was detected with ease.

SELECTIVE ACTION OF DYES AND OTHER DISINFECTANTS ON BACTERIOPHAGES. Alvin Y. Wells and Noble P. Sherwood. Jour. Inf. Dis., lv (1934), 2, p. 195.

A selective phagistatic action of gentian violet and crystal violet has been shown for the twelve bacteriophages tested. There appears to be a correlation between the selective action of the dyes on bacteria and that on their respective phages. The action of phenol on the bacteriophages seems to divide the lytic agents into two groups, as with the dyes, and is uniform. Sodium

hydroxide gives much more irregular results. Greater concentrations of phenol and sodium hydroxide were required to bring about inhibition of the phages than was necessary with the dyes. The phages which lyse Gram-negative bacteria are more resistant to deleterious substances than are those which lyse Gram-positive bacteria.

SALMONELLA AERTRYCKE VARIANT AS AN ETIOLOGIC AGENT OF PARATYPHOID IN PIGEONS. Erwin Jungherr and Kingston S. Wilcox. Jour. Inf. Dis., lv (1934), 3, p. 390.

A pigeon farm containing about 1,400 breeders suffered losses of from 15 to 20 per cent of the annual squab production. Although trichomoniasis and strongylosis seemed to be contributing factors, the isolation of a paratyphoid-like organism and the widespread sensitization to this organism in the breeding stock signified its etiologic importance. The causative organism produced acid and gas in broth containing dextrose and slight acid in dulcitol; it did not attack maltose, lactose and sucrose and was agglutinated by standard *S. pullorum* antiserum in dilutions of 1:1,200. The organism was identified as an atypical maltose-fermenting variant of *S. aertrycke*. Attention is called to the misleading influence of dissociation phenomena on cross-agglutination between *S. pullorum* and motile paratyphoid organisms and, in general, on the diagnosis of pullorum disease in unusual hosts.

CROSS-SPECIES TRANSMISSION STUDIES WITH DIFFERENT STRAINS OF BIRD-POX. Vernal Irons. Amer. Jour. Hyg., xx (1934), 2, p. 329.

The virus of pigeon-pox gave mild infection in the chicken. Its virulence for chickens was greatly enhanced on passage in the chicken. After a single passage in the chicken, the virus of pigeon-pox was greatly attenuated for the pigeon. Repeated passage, however, failed to destroy the infectivity for the pigeon. One strain of fowl-pox virus was transmissible with gradually increasing virulence in the pigeon but was temporarily attenuated for the chicken. Two other strains of fowl-pox virus were noninfectious for the pigeon. The chicken was susceptible but the pigeon was refractory to experimental turkey-pox. The turkey was susceptible to experimental fowl-pox. Ducks, guinea fowls and starlings were refractory to the strains of bird-pox. One strain of bird-pox (virus G of pigeon-pox) proved infectious

for the English sparrow and certain related species. Bird-pox virus was not pathogenic for small laboratory mammals; attempts to infect fetal rabbit by placental passage failed. While the chicken was susceptible to strains of vaccine virus, pigeons and English sparrows were refractory. The lesions of vaccinia and of bird-pox could readily be distinguished on the chicken by the inclusion bodies; vaccinia and fowl-pox failed to give cross-immunity on the chicken.

RESORPTION OF BACTERIA FROM THE GASTRO-INTESTINAL TRACT. Arvid Linden. Abst. Arch. Path., xviii (1934), 4, p. 562.

Behring, Ehrlich and others demonstrated that in newborn animals proteins and antitoxins pass unchanged from the gastro-intestinal tract into the blood. There is considerable disagreement about the question of resorption of microörganisms from the gastro-intestinal tract. Calmette's peroral vaccination against tuberculosis has awakened interest in this question. Linden gave India ink, carmine, timothy bacilli, bovine tubercle bacilli and B.C.G. to young mice and guinea pigs. Microscopic examination indicated that resorption of these substances and bacteria occurred rarely. The results of the study are not in agreement with Calmette's assumption of a general impregnation of the lymphoid system by the ingested bacteria.

PATHOLOGY OF THE CENTRAL NERVOUS SYSTEM IN CANINE BLACK TONGUE. Margaret Crane-Lillie and C. P. Rhoads. Arch. Path., xviii (1934), 4, p. 459.

Lesions were demonstrated by the Weigert stain in eleven of the twelve animals studied. The cytoplasm of the neuroglia cells in some instances were quite granular. In all the myelin stains the structural change was, for the most part, slight and consisted of irregularity, swelling and shrinking of the fibers. Of the twelve dogs, eleven showed alteration in the axis-cylinders by the Alzheimer-Mann stain; the axis-cylinders that were present in the lesions were swollen, broken up and tortuous, staining from purple through brilliant red. Poliomyelopathy, characterized by various types of degeneration in the nerve cells and the presence of fat in the cells, occurred in eleven of the twelve dogs. There was evidence of a general nervous disturbance of the structure of the central nervous system. The changes described appear to be an early acute type and perhaps autolytic or chemical.

Regular Army

Major Elwood L. Nye is, in addition to his other duties at the Presidio of San Francisco, Calif., detailed as port veterinarian, San Francisco port of embarkation, Fort Mason, Calif., and to duty at the headquarters of the 9th Corps Area.

The promotion of Lt. Col. Alfred L. Mason to the grade of colonel, with rank from November 13, 1934, is announced.

Second Lt. Curtis W. Betzold, now on duty at Carlisle Barracks, Pa., will report to the commandant, Medical Field Service School, that station, on or about December 29, 1934, for duty for the purpose of pursuing the basic course of instruction.

Second Lt. Wm. E. Jennings is relieved from further assignment and duty at Fort Leavenworth, Kan., and will proceed to Carlisle Barracks, Pa., and report to the commandant, Medical Field Service School, on or about December 29, 1934, for duty and for the purpose of pursuing the basic course of instruction.

Veterinary Reserve Corps

New Acceptances

Adan, Cirilo Lagmay (Filipino)	2nd Lt.	1123 N. Jefferson, Junction City, Kan.
Cox, John Leverton	2nd Lt.	2365 Warren St., Toledo, Ohio.
Ebert, Edgar Frazier	2nd Lt.	7230 Wornall Rd., Kansas City, Mo.
Groppe, Carl Wm	2nd Lt.	Stamms Lane, Wheeling, W. Va.
Orr, Norton Allison	2nd Lt.	616 S. Howes St., Fort Collins, Colo.
Siemer, Everett John	2nd Lt—	4524 Milwaukee St., Denver, Colo.

Promotions

	To	
Shaffer, Xenophon Bryan	Capt.	702 S. Richardson Ave., Vicksburg, Mich.
Starnes, Mervyn Benson	1st Lt.	304 City Hall Annex, Dallas, Tex.

Another Canine Hero

How Major, a dog in the home of Henry A. Spitzer, saved the family from death at the cost of his own life, is told in recent news dispatches from Saint Louis, Mo. Fumes from a leaky gas main filled the house as the family slept. Major awakened Mr. Spitzer, who called aid. An inhalator squad revived Mrs. Spitzer's mother and three children. Major was found dead in the basement.

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Final Report on Membership

It has been quite a task to complete the tabulation and classification of the membership in the Twelfth International Veterinary Congress. Quite a few applications for membership were received after the close of the Congress, many of these having been forwarded through national committees in different parts of the world, while the receipt of a few straggling applications was delayed for other reasons.

In the November issue of the JOURNAL was published what was believed to be a final report on the foreign membership. This did not include the 51 lady members enrolled but did include the ordinary, extraordinary and student members to the number of 755. The corrected figure, covering all classes of membership, is 888. Argentina was responsible for the largest part of the difference between the figures in the two reports, 67 applications having been received since the first report was compiled. Argentina should be credited with a total of 69 members, placing this country in third place, Germany having been first and Great Britain and Northern Ireland, second.

The following table shows the distribution of the total membership of 3,917:

Ordinary members:	Foreign	813	
	U. S. A.	2,441	
			3,254
Extraordinary members:	Foreign	22	
	U. S. A.	51	
			73
Student members:	Foreign	2	
	U. S. A.	63	
			65
Lady members:	Foreign	51	
	U. S. A.	474	
			525
		Grand total	3,917

The distribution of the 2,441 ordinary members for the United States is shown in the following table. It will be noted that New York led the states by a wide margin, having a total of 429 ordinary members enrolled. California and Pennsylvania ran a nice race for second place, the Golden State coming out a winner over

the Keystone State by the small margin of four members. Ohio had fourth place, New Jersey fifth, Illinois sixth and Minnesota seventh, all of the states named having been credited with more than 100 members.

Alabama	9	Nevada	13
Arizona	11	New Hampshire	25
Arkansas	1	New Jersey	119
California	185	New Mexico	4
Canal Zone	1	New York	429
Colorado	48	North Carolina	19
Connecticut	39	North Dakota	12
Delaware	5	Ohio	132
District of Columbia	68	Oklahoma	53
Florida	26	Oregon	20
Georgia	10	Pennsylvania	181
Hawaii	4	Puerto Rico	4
Idaho	4	Rhode Island	11
Illinois	105	South Carolina	10
Indiana	45	South Dakota	11
Iowa	89	Tennessee	17
Kansas	40	Texas	40
Kentucky	12	Utah	8
Louisiana	15	Vermont	30
Maine	16	Virginia	27
Maryland	36	Virgin Islands	1
Massachusetts	79	Washington	30
Michigan	34	West Virginia	15
Minnesota	103	Wisconsin	92
Mississippi	12	Wyoming	9
Missouri	74		
Montana	9		
Nebraska	49	Total	2,441

University of Nebraska Held to Be Wholesaler

A permanent injunction was issued by Federal Judge Philip L. Sullivan, in Chicago, on December 21, 1934, restraining the Aurora Serum Company, of Aurora, Ill., from fulfilling a contract with the University of Nebraska, because of an alleged price violation under the NRA Code for the Anti-Hog Cholera Serum and Hog Cholera Virus Industry. The Aurora Serum Company, according to report, had contracted to sell 6,000,000 cc of anti-hog cholera serum to the University of Nebraska at 27 cents per 100 cc. Assistant U. S. Attorney Austin Hall charged that the serum should be sold to the University of Nebraska as a wholesaler, at the code price of 34 cents per 100 cc. The attorney for the serum company held that the University of Nebraska was entitled to the lower price, in view of the fact that it makes serum in its own laboratory and, for that reason, is a producer.

Standards for Canned Dog Food

The Code Authority for the Dog Food Industry, through its Executive Officer and Counsel, Charles Wesley Dunn, recently released for publication "The Definitions and Standards of Identity and Biological Value and Labeling Requirements for Canned Dog Food and a Plan for Their Enforcement." The Code of Fair Competition for this industry under the NRA made mandatory the setting up of these standards and in this respect this particular code was unique as compared with those of other industries for which codes had been promulgated.

This infant industry has grown rapidly since its inception about ten years ago and, during the past six months, has pioneered along the most progressive lines to correct abuses that were tending to destroy the good will of many consumers, which a few manufacturers had built up through large expenditures for advertising and investments in modern factories and equipment.

Seeing the need for the establishment of a basic and fair biological value for dog food, the Code Authority for the industry has taken full advantage of the opportunity presented under the NRA and has suggested standards which offer to the buyers of dog food safeguards that have not been obtainable previously. This constructive effort is the key to the success of the whole reform movement in this industry.

The plan of enforcement of the provisions of this report calls for the setting up by the Code Authority for the Dog Food Industry of a "Scientific Council for the Dog Food Industry." This Council of five members, none of whom shall have any interest in the dog food industry, shall consist of an experienced authority on canine nutrition, who shall be chairman; an expert in the general field of nutrition; an expert in food chemistry; a representative of the veterinary medical profession who is affiliated with an accredited American veterinary college; and a representative of the United States Government, or, if such an individual is not available, a suitable representative of the public at large.

This Council will act as a scientific referee and research agency

in an advisory capacity to the Code Authority. It will investigate scientific questions relating to the manufacture of dog foods, their chemical combination and composition; prescribe standards and methods; determine when violations exist; report on compliance and regulations necessary to effectuate compliance with the standards adopted and approved by the Code Authority.

When the Council reports favorably on any canned dog food, then that manufacturer will have the right to use a legend reading, "Approved by the Scientific Council for the Dog Food Industry," on the packages and in the advertising of this product approved under such terms and conditions as the Council may prescribe.

A fully equipped control laboratory will be established for the Council in one of the leading eastern universities, where the work of the Council will be carried on in a scientific atmosphere of the highest type.

This wholehearted effort by this new industry is of utmost importance at this time and indicates the new spirit of unselfish public duty that prominent business men follow in the fulfillment of a new order of ethics in the business life of our country.

The National Dog Food Manufacturers' Association, which originally sponsored a Code of Fair Competition under the NRA through its officers, directors and members, has supported Charles Wesley Dunn, their general counsel, in his unremitting efforts to place the industry in a high position in the confidence of the public. Mr. Dunn for more than 25 years has been identified with the movement for progressive legislation on all matters affecting the public interest in the food and drug fields. He has been foremost in championing a new federal food and drug act. It is believed that, in his report on the Standards of Identity and Biological Value for Canned Dog Food, Mr. Dunn has written basic law that will be a strong foundation for the future growth of the industry.

Doctor Steddom Retired

The retirement on December 31, 1934, of Dr. R. P. Steddom, Chief of the Meat Inspection Division of the U. S. Bureau of Animal Industry, marked the termination of 37 years of active service. During this time, Dr. Steddom supervised the upbuilding of a service which now inspects annually more than 75,000,000 food animals before and after slaughter and has won worldwide recognition for its high scientific standards, practical bene-

fits to industry, and protection to public health. The service has consistently kept abreast of commercial developments and in many cases has made distinct contributions to improvements in equipment and methods.

In commending Dr. Steddom at the close of his distinguished career as a public officer, Dr. John R. Mohler, Chief of the Bureau of Animal Industry, paid tribute to his administrative capacity as well as to the evidence of his scientific ability. "Your genius has expressed itself," Dr. Mohler stated, "in the selection and training of capable assistants and associates, with the result that the ideals and standards which you have established will be perpetuated for many years to come."

DR. R. P. STEDDOM

A native of Ohio, Dr. Steddom was graduated from the Ontario Veterinary College in 1886, and engaged in veterinary practice in Iowa and Illinois until 1897, when he was appointed to the Bureau as assistant inspector. In the early years of his service, he was stationed in Kansas City, Mo., and traveled throughout the southwestern part of the United States and in Puerto Rico. Later, as chief of the meat inspection service, his official station was in Washington, D. C.

Dr. Steddom joined the American Veterinary Medical Association in 1892 and enjoys the distinction of a longer period of active membership in the national organization than any other veterinarian connected with the U. S. Bureau of Animal Industry, past or present.

Royal Veterinary College Appeals for Aid

The Royal Veterinary College of London took part in the Lord Mayor's Show on November 9, 1934, and gave a good account of itself in the splendid program it presented. This is the second time the College has been enabled to press its claims for the support of the citizens of London through the medium of the show, according to the *Veterinary Record.*

That part of the show given by the College was headed by a banner with the words: "Help to make the Royal Veterinary College worthy of your city. Founded 1791; rebuilding 1934." This was followed by a car showing an animal operating theater with students and nurses. Next came four veteran war horses; then a car depicting the world's largest nosebag gleaning for the 250,000,000 Farthing Fund in aid of the College. Close on this was shown "Ye Olde English Forge" in full blast; and last came animal patients and their nurses. Each car was drawn by a team of four Suffolk Punch horses.

Cleveland Veterinarians Operate on Tigress

When Susie, a nine-year-old tigress in the Brookside Park Zoo, Cleveland, Ohio, was attempting unsuccessfully to present the Zoo with tiger cubs, on November 28, 1934, three Cleveland veterinarians were called in: Drs. Harry Roberts (O. S. U. '33), Clifford Wagner (O. S. U. '26) and K. K. Goekdjian (Colo. '30). They set to work. The first cub was born dead. The second, also dead, came two hours later. In a desperate effort to save the mother's life, the cage was turned into an operating-room. A local anesthetic was administered while Susie, usually a ferocious animal, lay quietly as the veterinarians performed a Cesarean operation. Forty minutes later, a third cub was delivered, dead. There was little hope that Susie, weakened by her ordeal, could pull through.

Short on Bovine Anatomy

"Now tell the jury, lady," instructed the young lawyer, "just where the prisoner was milking the cow."

The young lady, a trifle embarrassed, smiled sweetly and replied, "Why I think it was a little back of the center, sir."

California Dairyman.

SOUTHERN STATES VETERINARY MEDICAL ASSOCIATION

The nineteenth annual meeting of the Southern States Veterinary Medical Association was held in conjunction with the annual meeting of the Florida Veterinary Medical Association, at the Mayflower Hotel, Jacksonville, Fla., October 29-30, 1934. Dr. R. S. MacKellar, president of the A. V. M. A., accompanied by Mrs. MacKellar, was the guest of honor.

The first-day session was opened with an invocation by Rev. Albert J. Kissling, pastor of the Riverside Presbyterian Church. The address of welcome was given by Mayor John T. Alsop, with a response by Dr. L. A. Mosher, of Atlanta, Ga. Following the President's address by Dr. H. C. Nichols, of Ocala, Fla., two papers were presented: "Tick Paralysis Clinically," by Dr. John R. Wells, of West Palm Beach, Fla., and "*Crotalaria Spectabilis* Poisoning of Domestic Animals," by Drs. D. A. Sanders and M. W. Emmel, of the Florida Agricultural Experiment Station.

In the afternoon, Dr. R. S. MacKellar addressed the Association on the subject of the A. V. M. A. The papers presented included: "The Chemical Process of Branding," by Dr. E. M. Nighbert, of Cantonment, Fla.; "Screw-Worm Infection of Live Stock," by Dr. W. E. Dove, of the U. S. Bureau of Entomology, Atlanta, Ga.; "Large-Animal Surgery," by Dr. W. F. Guard, of Ohio State University. Dr. Dove's paper was discussed by Drs. W. E. White, chief veterinarian of the Georgia Department of Agriculture, and H. B. Raffensperger, of the U. S. Bureau of Animal Industry, Moultrie, Ga.

In the evening, the annual banquet was held, with Dr. J. V. Knapp, state veterinarian of Florida, acting as toastmaster. Following the recognition of prominent guests, the address of the evening was given by Mr. L. M. Rhodes, chief of the Florida State Marketing Bureau. A dance closed the evening's festivities.

The second-day program was opened with a splendid paper by Dr. S. H. Regenos, of Zionsville, Ind., on "Canine Distemper and

Its Control." Other interesting papers were given as follows: "Veterinary Proprietary Remedies and the Food and Drug Act," by Dr. H. E. Moskey, of the Food and Drug Administration, Washington, D. C.; "Some Important Poultry Disease Problems Confronting the Veterinarian," by Dr. Fred D. Patterson, of the Alabama Polytechnic Institute; "My Experiences in an Effort to Eliminate Doping in Horses," by Dr. J. G. Catlett, of Miami, Fla.; "The Etiology of Fowl Paralysis and Leucosis," by Dr. M. W. Emmel, of Gainesville, Fla.

Several of the visitors took advantage of the occasion to make side trips to other parts of Florida, Saint Augustine being of special interest. The ladies were entertained with a bridge party, a sight-seeing trip and a shopping tour.

Officers of the Southern States Veterinary Medical Association for 1935 were chosen as follows: President, Dr. G. R. Kitchen, Sumter, S. C.; first vice-president, Dr. L. J. Kepp, Atlanta, Ga.; second vice-president, Dr. John H. Gillmann, Memphis, Tenn.; secretary-treasurer, Dr. M. R. Blackstock, Spartanburg, S. C. reëlected).

M. R. BLACKSTOCK, *Secretary.*

EAST TENNESSEE VETERINARY MEDICAL SOCIETY

The quarterly meeting of the East Tennessee Veterinary Medical Society was held at Knoxville, December 8, 1934. Veterinarians from all over East Tennessee attended the meeting, which was one of the most interesting the Society has held. Of special interest was a discussion of the eradication of Bang's disease, by Drs. W. B. Lincoln, of Morristown, R. E. Bauch, of Knoxville, and W. A. Jones, of Johnson City. Officers reëlected to serve during the coming year are: President, Dr. W. B. Lincoln, Morristown; secretary, Dr. R. E. Baker, Morristown; treasurer, Dr. C. D. White, Knoxville.

NORTHEASTERN INDIANA VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Northeastern Indiana Veterinary Medical Association was held at the Catholic Community Center, Fort Wayne, December 11, 1934. Dr. Victor H. Hilgemann, of Fort Wayne, was the speaker of the evening, his address being

illustrated with moving-pictures of big game hunting in Canada. Officers elected for the coming year are: President, Dr. F. A. Hall, Garrett; vice-president, Dr. L. P. Meyer, Convoy, Ohio; secretary-treasurer, Dr. H. O. Elliott, Orland.

SOUTHERN OHIO VETERINARY MEDICAL ASSOCIATION

The Southern Ohio Veterinary Medical Association held its annual fall meeting at the Cherry Hotel, Washington Court House, December 13, 1934, with 29 members from eleven counties in attendance. The meeting, which took the form of a banquet, was thoroughly enjoyed. The speaker of the evening was Dr. P. T. Engard, of Marysville, whose address on "Poultry Practice," proved unusually interesting, and provoked a lively discussion. Dr. J. A. McCoy, of Washington Court House, president of the Association, presided during the meeting.

WABASH VALLEY VETERINARY MEDICAL ASSOCIATION

The Wabash Valley Veterinary Medical Association held its quarterly meeting at the Hotel Spencer, Marion, Ind., December 19, 1934, with 20 veterinarians from several counties in attendance. Speakers on the program were Dr. R. E. Smith, of Boswell, Ind., who spoke on "Forage Poisoning," and Dr. R. J. Hoskins, of Indianapolis, Ind., who discussed "Small-Animal Practice." A dinner followed the meeting. Eight wives who accompanied their husbands were entertained by Mrs. G. E. Botkin, of Marion, at a bridge party at the hotel. Officers were reelected as follows: President, Dr. S. F. Gaynor, Logansport, Ind.; vice-president, Dr. J. H. Mills, Russiaville, Ind.; secretary-treasurer, Dr. R. G. Fellers, Walton, Ind.

Must Prove Right to Secure Hunting License

To get a hunting license in Holland, a hunter must prove that he has bettered game conditions through the year; that he has raised birds and released them, fed them during the winter or, in some other way, encouraged the increase of wild life. When he can do this, he is permitted to buy a license for which he must pay $20.

Animaldom.

NECROLOGY

THEOBALD SMITH

Dr. Theobald Smith, president of the Board of Scientific Directors of the Rockefeller Institute for Medical Research and, from 1915 to 1929, director of the Department of Animal Pathology, at Princeton, N. J., died December 10, 1934, of heart disease. He had been a hospital patient just prior to his death for a malignant growth in the intestines. Dr. Smith was an honorary member of the American Veterinary Medical Association, having received that honor in 1897 after having been an active member of the Association for five years.

Born in Albany, N. Y., July 31, 1859, Dr. Smith studied at Cornell University and later entered the Albany Medical College, from which he received his medical deyree in 1883. One year later, he entered the service of the U. S. Bureau of Animal Industry and, for ten years, was engaged in studying infectious diseases of animals. It was while he was engaged in this work, coöperating with Drs. Cooper Curtice and F. L. Kilborne, that the epochal discovery was made of the rôle of the cattle tick in its relation to southern cattle fever.

In 1895, Dr. Smith was appointed Professor of Applied Zoölogy at Harvard University, and later he became Professor of Comparative Pathology. He served in these positions until 1914, at the same time directing the antitoxin and vaccine laboratory of the Massachusetts Board of Health, the first state organization of the kind to be created. He served as a director of the Carnegie Institution, and was a scientific director of the Rockefeller Institute for Medical Research from its beginning, later becoming vice-president and, in 1933, on the death of Dr. William H. Welch, president.

In recognition of his services to science and to medicine, Dr. Smith was honored by universities and medical societies throughout the world. He received the A. M. degree from Harvard University in 1901, and the Sc. D. from the same institution in 1910. The University of Chicago honored him with the LL.D. degree in 1907, and Washington University similarly honored him in

1915. Both Yale University and Princeton University conferred the Sc. D. degree in 1917. He was given the degree, Doctor of Veterinary Medicine *(causa honoris)*, by the Royal Hungarian Veterinary College, and was an honorary member of the Royal Society of Medicine, of London. In 1933, the Society further honored Dr. Smith by awarding him the Copley Medal, for his original research and observation on diseases of animals and men.

Dr. Smith is survived by his widow (née Lillian Hillyer Egleston), two daughters and one son.

WALTER A. DAVIDSON

Dr. Walter A. Davidson, of Chicago, Ill., died March 1, 1934, from heart disease, following an illness of about a year. Born in Indianapolis, Ind., November 4, 1875, Dr. Davidson was graduated from the Kansas City Veterinary College in 1908. He joined the U. S. Bureau of Animal Industry, October 26, 1908, and served in the positions of veterinary inspector and assistant veterinarian from that date until his retirement, June 30, 1932. During the entire period of his connection with the B. A. I., he was stationed at Chicago. Dr. Davidson joined the A. V. M. A. in 1919. He is survived by his widow.

EDWARD THOMSON FRANK

Dr. E. T. Frank, of Warren, Minn., died March 20, 1934.

Born at Omro, Wis., March 19, 1867, Dr. Frank removed to Warren with his parents in 1882. He attended the Warren public schools, Moorhead Teachers' College and the Dixon (Ill.) Business College. He then entered the Chicago Veterinary College and was graduated in 1897. Following his graduation, he returned to Warren and practiced there until his death. In 1904, he left his practice long enough to take a postgraduate course at the Kansas City Veterinary College.

Dr. Frank was a member of the Odd Fellows Lodge, being a past Noble Grand of the local order. He took great interest in organizing the Marshall County Agricultural Society, serving as secretary of the board of directors until illness prevented his carrying on the active duties of the office. He was a lover of good horses and always kept a stable of Thoroughbreds.

Surviving Dr. Frank are his widow (née Carrie Carlson) a son, a daughter, one brother and two sisters.

J. P. F.

HANNES A. HELA

Dr. H. A. Hela, of Menahga, Minn., died suddenly April 9, 1934, at 66 years of age. He attended the Ontario Veterinary College for one session (1893-94) before entering the Chicago Veterinary College, from which he was graduated in 1900. Following his graduation, Dr. Hela located at Cokato, Minn., and later entered the service of the U. S. Bureau of Animal industry. He was first stationed in Chicago, Ill., and from there was transferred to Milwaukee, Wis. Resigning from government service in 1909, he located in Granite Falls, Minn., and later removed to Wadena, Minn., where he practiced for several years.

Dr. Hela was a member of the Board of County Commissioners of Wadena County at the time of his death, and, at the risk of his own political fortunes, was largely instrumental in obtaining an area tuberculin test for that county. He was highly esteemed in the community. He is survived by his widow.

J. P. F.

NATHAN H. DOWNS

Dr. Nathan H. Downs, of Los Angeles, Calif., died May 4, 1934, following an illness of six days with acute enteritis. He was 59 years of age. Dr. Downs had just returned from a trip around the world when he was taken ill. Dr. Downs was reared in Geneva, Ohio, and was well known there. He was a graduate of the Chicago Veterinary College, class of 1893.

THOMAS W. VANCURA

Dr. T. W. Vancura, of New Prague, Minn., died at his home, July 13, 1934, following a heart attack. He was stricken and died within an hour, following a day during which he had gone about his duties as usual.

Born in Lakefield, Minn., November 10, 1892, he attended high school at Lakefield. Later, he was graduated from the School of Agriculture at the University of Minnesota before he decided to enter the veterinary profession. He attended the McKillip Veterinary College and won his degree in 1916. He served with the Veterinary Corps during the World War, and was stationed at the Veterinary Training School, Camp Lee, Va. He located in New Prague in 1917 and continued in practice there until his death.

Dr. Vancura joined the A. V. M. A. in 1928. He was president of the Minnesota Live Stock Show, and past-president of the 40-8 Voiture of New Prague. He was a fourth-degree member of Council 1923, Knights of Columbus; a member of Charles Borak Post of the American Legion; of the New Prague Fire Department, the New Prague Community Club, the Community Sportsmen's Club and the Last Man's Club. He was a director of the Legion Pavilion Corporation and a member of the local Boy Scout committee. He was the first member of the Last Man's Club to die. During the hour of his funeral, flags were at half mast in deference to his memory.

Surviving Dr. Vancura are his widow (née Bessie A. Dobihal), three sons, one daughter, his mother, five brothers and four sisters.

J. P. F.

WILLIAM GETTY

Dr. William Getty, of Mapleton, Minn., died July 22, 1934, following an illness of six weeks of a heart ailment.

Born in Beauford Township, Minn., December 29, 1876, Dr. Getty grew to manhood on the home farm, attending the school of his district. In 1899, he became a barber and located in Easton, Minn. Later, he purchased a shop at Minnesota Lake and remained there several years. He then decided to study veterinary medicine and matriculated at the Kansas City Veterinary College, from which he was graduated with honors in 1916. Returning to Minnesota to practice, Dr. Getty located at Amboy, where he established a hospital and built up a splendid practice. In 1925, he sold his practice in Amboy and located at Mapleton, where he entered whole-heartedly into the life of the village. He was active in civic affairs and served for two years as president of the Commercial Club.

Surviving are his widow (née Emma Tolzmann), one daughter, and four brothers.

J. P. F.

JAMES H. MARTIN

Dr. J. H. Martin, of Spokane, Wash., died in Portland, Ore., October 18, 1934. He was a graduate of Washington State College, class of 1910, and had been deputy state veterinarian of Washington for many years prior to his death. As deputy state

veterinarian, his work was concerned primarily with tuberculosis eradication.

E. E. W.

F. ALFRED KRETSCH

Dr. F. A. Kretsch, of Fairfax, Minn., died October 18, 1934, as the result of an automobile accident. Dr. Kretsch was injured, September 23, when he was caught between the front and rear bumpers of two cars, as he stood behind his car, preparing to have it towed to town. Both legs were broken and, already suffering from a weakened heart, he was unable to withstand the shock.

Born in New Ulm, Minn., August 15, 1887, Dr. Kretsch was graduated from the New Ulm High School in 1906. After working as a messenger for a short time, he entered the Chicago Veterinary College and received his degree in 1911. Immediately following his graduation, he located at Fairfax and engaged in general practice which he continued up to the time of his death, with the exception of a period spent in the Veterinary Corps during the World War. He joined the Medical Enlisted Reserve Corps, June 12, 1918. Several months later he was transferred to the Veterinary Corps and ordered to active service at Camp Lee, Va., where he remained until the Armistice, when he was honorably discharged and returned to Fairfax.

Dr. Kretsch was a member of the Knights of Columbus and of Saint Joseph's Society. He was a charter member of the American Legion, B. B. M. Post, and was its commander for several years. He was a member of Gamma Chapter of Alpha Psi Fraternity. Surviving are his widow (née Martha Maud Kipp), one daughter, one brother and two sisters.

J. P. F.

JOHN McCARTNEY

Dr. John McCartney, of Middletown, N. Y., died at his home, November 2, 1934, following an illness of about two weeks, brought on by a stroke of apoplexy. He was apparently recovering when a second stroke occurred and he died within a few hours. Dr. McCartney had enjoyed excellent health up to the time of his final illness.

Born in Coldenham, N. Y., Dr. McCartney was graduated from the New York State Veterinary College, Cornell University, in 1909. He located in Middletown in 1912 and, for the past 20

years, had occupied the position of field veterinarian of Borden's Farm Products Company, Inc. Dr. McCartney was well thought of in his community, as evidenced by the throngs from all walks of life who attended his funeral.

Dr. McCartney joined the A. V. M. A. in 1911. He was a past-president of the Hudson Valley Veterinary Medical Society and a member of the New York State Veterinary Medical Society. He was an elder of the First Presbyterian Church and a past-commander of Cypress Commandery. Memberships in other organizations included: Walden Lodge, F. & A. M.; Midland Chapter 240, R. A. M.; Mecca Temple, Mystic Shrine; the Excelsior Hook and Ladder Company and the Middletown Club. Surviving are his widow (née Anna Corwin) and a sister.

I. O. D.

A. E. BEHNKE

Dr. A. E. Behnke, of Milwaukee, Wis., died November 12, 1934, after an illness of about one month. The cause of death was given as carcinoma of the liver secondary to carcinoma of the sigmoid colon.

After spending his early life at New Ulm, Minn., Dr. Behnke entered the Chicago Veterinary College and was graduated with the class of 1892. He entered the service of the U. S. Bureau of Animal Industry, October 1, 1895, following his appointment as assistant inspector, and was assigned to meat inspection in Chicago. He was transferred to South Saint Paul, Minn., as inspector in charge, effective June 15, 1896, and from there to Cudahy and Milwaukee, Wis., in charge June 15, 1897. He was promoted and, on December 1, 1906, transferred to Washington, D. C., where he became associate chief of the Meat Inspection Division until September 16, 1912, when he returned to Milwaukee as inspector in charge. On April 19, 1926, he was transferred to Sioux City, Iowa, as inspector in charge, where he remained until June 22, 1928, when he returned to Milwaukee and remained until his retirement at the close of December, 1932.

Dr. Behnke joined the A. V. M. A. in 1898. He was a member of the Special Committee on Schmidt Memorial (1925-26) and a member of the special committee (1931-32) appointed to study the presidential address of Dr. Maurice C. Hall. He was a member of the National Association of B. A. I. Veterinarians, of the United States Live Stock Sanitary Association and of the Twelfth International Veterinary Congress.

WALTER RICHARD DONAHOE

Dr. Walter R. Donahoe, of Sauquoit, N. Y., died November 30, 1934. He was a graduate of the New York State Veterinary College, at Cornell University, class of 1923, and was located at Clinton, N. Y., before going to Sauquoit.

ROBERT M. THOMPSON

Dr. Robert M. Thompson, of Darlington, Wis., died December 2, 1934, following an automobile accident. Dr. Thompson, who had spent Thanksgiving with relatives in Waukesha, Wis., was returning home when his automobile skidded off the icy highway. He was uninjured and was at the scene of the accident waiting for the wrecker when he was struck by a skidding truck and fatally injured. Dr. Thompson, who was 69 years old, was a graduate of the Ontario Veterinary College, class of 1889.

JOHN L. REDDING

Dr. John L. Redding, of Waukegan, Ill., died at his home, December 11, 1934, after an illness of several weeks. Congestion of the kidneys was reported as the cause of his death. He was 41 years of age.

Born on the old Redding farm in Benton Township, Ill., Dr. Redding received his early education in the Waukegan public schools, later attending the Waukegan Business College. He then matriculated at the Chicago Veterinary College and was graduated in 1913, at the age of 20 years. He located in Waukegan, where he established a lucrative practice throughout Lake County, Ill., and Kenosha County, Wis. Dr. Redding was a member of Antioch Lodge, A. F. & A. M., at Milburn, Ill., and also a member of the O. E. S. Surviving are one son, his mother, one sister and one brother.

McCLINTIC NEFF

Dr. McClintic Neff, of Lexington, Va., was killed in an automobile accident on December 17, 1934. Dr. Neff was a graduate of the Ontario Veterinary College, class of 1932, and had established a practice in Lexington. He was the son of Dr. S. C. Neff (Ont. '01), of Staunton, Va.

I. D. W.

EDMOND EVERETT BITTLES

Dr. E. E. Bittles, of Waterford, Pa., died suddenly, December 19, 1934, at Venus, Pa., while he and Mrs. Bittles were en route to Harrisburg to visit his sisters.

Born at Waterford, October 12, 1863, Dr. Bittles received his early education in the township school and the Waterford Academy. He entered the Ontario Veterinary College and was graduated in 1890. He located first in Greenville, Pa., where he practiced for three years. He then removed to Union City, Pa., and remained there for three years before locating in New Castle, Pa., where he practiced until 1917. For many years, Dr. Bittles and Dr. E. C. Porter (Ont. '91) practiced very harmoniously together in New Castle. In 1917, Dr. Bittles became associated with the late Dr. S. R. Craver, of Youngstown, Ohio. After two years, in 1919, he retired from active practice and returned to Waterford to make his home. Dr. Bittles invented several veterinary instruments which were widely used by the profession and from which he never asked or received any royalties. Many veterinarians from western Pennsylvania and eastern Ohio attended the funeral services, and four of the six pallbearers were veterinarians: Dr. Earle S. Pickup, of Union City, Pa.; Dr. Eugene M. Coover, of Erie, Pa.; Dr. Nevin S. Craver, of Youngstown, Ohio, and Dr. M. Park Hendrick, of Meadville, Pa.

Dr. Bittles joined the A. V. M. A. in 1923. He was also a member of the Pennsylvania State Veterinary Medical Association and served one year (1922-23) as its president. He was a member of the Northwestern Pennsylvania Veterinary Club and of the Twelfth International Veterinary Congress. He was a member of the Pennsylvania State Board of Veterinary Medical Examiners at the time of his death, having served as president of the Board for one year. He was elected to the State Legislature from Lawrence County in 1912, and was an intimate friend of Governor Gifford Pinchot for many years.

Surviving Dr. Bittles are his widow (née Belle Hinwood), one brother and three sisters.

B. H. B.

Our sympathy goes out to Dr. E. W. Holden (Corn. '34), of Route 2, Norristown, Pa., in the death of his wife who died, November 27, 1934, as the result of a brain tumor.

Prosperity makes friends, adversity tries them.—PUBLIUS SYRUS.

PERSONALS

MARRIAGES

DR. GERARD B. MERRICK (O. S. U. '31), of Sandusky, Ohio, to Miss Manon Trei, of Forreston, Ill., at Lowell, Ind., August 13, 1934.

DR. K. L. RITCHIE (Iowa '33), of Cleghorn, Iowa, to Miss Esther Sather, of Roland, Iowa, September 29, 1934, at Roland.

DR. WILLIAM JOHN BOYD (U. P. '33), of Sewickley, Pa., to Miss Virginia Morton Findlay, at Swarthmore, Pa., October 20, 1934.

DR. KENNETH BURRISS (O. S. U. '31), of New Bedford, Mass., to Miss Margaret Diven, of Hillsboro, Ohio, December 22, 1934, at Columbus, Ohio.

DR. N. L. SIPLOCK (Ont. '31), of Chardon, Ohio, to Miss Harriet McCray, of Ashland, Ohio, December 24, 1934, at Ashland.

DR. O. B. CURRY (O. S. U. '25), of Morristown, Ind., to Miss Ruth Rafferty, of Morristown, December 26, 1934, at Morristown.

BIRTHS

To DR. and MRS. L. L. DUNN, of Dell Rapids, S. Dak., a daughter, Beverly Jeanne, September 28, 1934.

To DR. and MRS. J. C. LUCKEROTH, of Seneca, Kan., a daughter, Mary Lou, September 30, 1934.

To DR. and MRS. JAY G. TOWNSEND, of Los Angeles, Calif., a son, Jay Hinze, November 29, 1934.

PERSONALS

DR. J. H. HOGAN (McK. '09) has removed from South San Francisco to Burlingame, Calif.

DR. LEO M. STECKEL (O. S. U. '07) is spending the winter at Wintergarden, Palm Springs, Calif.

DR. W. L. BOLIN (St. Jos. '18-Ont. '21), formerly of Turtle Lake, Wis., is now located in Chicago, Ill.

DR. F. D. WEIMER (K. C. V. C. '16) has changed locations from Cumberland, Iowa, to Anita, same state.

DR. J. S. GROVE (Ont. '92) has removed from Fort Worth, Tex., to Dallas. Address: 2126 Maryland Avenue.

DR. J. E. MCDERMID (O. S. U. '03), of Ladysmith, Wis., was elected to the State Legislature at the fall elections.

DR. EDGAR D. WRIGHT (Ont. '21), of West Mansfield, Ohio, reports a change of address to Bluffton, Ind., R. F. D.

DR. EDWARD C. JESPERSEN (McK. '16), formerly at Fort Atkinson, Wis., is now located at Stevens Point, same state.

DR. PERCY M. ALDRICH (Wash. '32) has reported a change of address from Spokane, Wash., to Wenatchee, Wash., Route 5.

DR. CHARLES H. HIGGINS (McGill '96), of New York, N. Y., is president of the New York Veteran Druggists' Association.

DR. GEO. C. FAVILLE (Iowa '80), who has been located in Richmond, Va., for the past several years, has removed to Emporia, Va.

DR. H. E. BEARSS (Chi. '09), of Minonk, Ill., has built a small-animal hospital on the rear of his residence lot on North Chestnut Street.

DR. JOSEPH S. BARBER (Chi. '10), of Central Falls, R. I., was confined to his home for three weeks recently with an attack of the grippe.

DR. FORREST MCCLEAD (O. S. U. '33), formerly of Coatesville, Ind., has joined the staff of the Ellin Prince Speyer Hospital, New York, N. Y.

DR. WILLIAM JOHN BOYD (U. P. '33) is now associated in general practice with his father, Dr. Charles W. Boyd (U. P. '95), at Sewickley, Pa.

DR. ALEXANDER FINDLAY (Ont. '91), of Mission City, B. C., has been appointed a stipendiary magistrate for the County of New Westminster, B. C.

DR. W. WALTER MARTIN (U. P. '95), of Spring Lake, N. J., has gone to Florida for the winter. His winter residence is at 217 S. E. 14th Street, Miami.

DR. ELMER W. BABSON (Harv. '97) and his son, Dr. Osman Babson (Corn. '34), of Gloucester, Mass., are planning to build a small-animal hospital in the spring.

DR. F. H. MCCLEAN (T. H. '13), of Griggsville, Ill., has accepted an appointment in the U. S. B. A. I., and was ordered to report at Tupelo, Miss., early in December.

DR. R. M. PRITCHARD (Wash. '30), of Tacoma, Wash., is looking after the practice of Dr. E. F. McCune (Wash. '31), who is temporarily in the service of the U. S. B. A. I.

DR. E. E. BOLES (Ind. '13), of Warsaw, Ind., was the speaker of the day at the meeting of the local Kiwanis Club, November 15. His topic was "The Veterinary Profession."

DR. THOMAS P. GALLAHUE (Chi. '17), of Waukegan, Ill., has been retained another year as Lake County Veterinarian by a unanimous vote of the County Board of Supervisors.

DR. A. C. JERSTAD (Wash. '33), formerly with the Puritan Poultry Corporation, at Atascadero, Calif., is now at the Western Washington Experiment Station, Puyallup, Wash.

DR. C. D. LOGSDON (Mich. '34) has located for general practice at Perry, Mich., following a temporary U. S. B. A. I. appointment of three months, on meat inspection, in Chicago, Ill.

DR. G. W. RAWSON (U. S. C. V. S. '16), of Detroit, Mich., is the author of an article in the *Scientific Monthly* for December, 1934, on "Arthropods and Their Relationship to Diseases of Domestic Animals."

DR. P. P. FORSBERG (Chi. '14), of Hobart, Ind., has been appointed Lake County Veterinarian, succeeding Dr. F. G. Roth (Ind. '17), of Crown Point, Ind., who had held the office for the past fourteen years.

DR. M. R. BLACKSTOCK (Colo. '10), of Spartanburg, S. C., secretary of the Southern States Veterinary Medical Association, sustained a broken ankle early in November, and was incapacitated for several weeks.

DR. E. F. THOMAS (Ga. '26), who was formerly connected with the Florida Agricultural Experiment Station at Gainesville, has removed to Ocala, Fla., where he is associated in practice with Dr. H. C. Nichols (O. S. U. '17).

DR. E. F. MCCUNE (Wash. '31), formerly of Tacoma, Wash., has accepted a temporary appointment with the U. S. B. A. I., and is now engaged in tuberculosis eradication in Cowlitz County, Wash. His address is Route 1, Kelso, Wash.